A STUDY OF PATIENTS PROFILE WITH ACUTE SYMPTOMATIC SEIZURES IN ANNAL GANDHI MEMORIAL GOVERNMENT HOSPITAL, TRICHY

Dissertation submitted to
THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
in partial fulfillment of the regulations
for the award of the degree of
M.D. BRANCH – I

GENERAL MEDICINE
K.A.P.V. GOVERNMENT MEDICAL COLLEGE, TIRUCHIRAPPALLI
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI
APRIL – 2012

CERTIFICATE

This is to certify that the dissertation entitled "A STUDY OF PATIENTS PROFILE WITH ACUTE SYMPTOMATIC SEIZURES IN ANNAL GANDHI MEMORIAL GOVERNMENT HOSPITAL,TRICHY" is the bonafide original work of **Dr. ABDUL REHAMAN** in partial fulfillment of the requirements for M.D. Branch-I (General Medicine) Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in April 2012.

Prof. Dr.V. RAJENDRAN M.D.,
Associate Professor of Medicine,
Unit Chief,
K.A.P.V. Govt. Medical College,
Trichy.

Prof. Dr.C. ASOK KUMAR M.D.,
Professor & Head of the
Department of Medicine,
K.A.P.V. Govt. Medical College,
Trichy.

Prof. Dr. A.KARTHIKEYAN,M.D.,
DEAN
K.A.P.V. Govt. Medical College,
Trichy .

DECLARATION

I **Dr. ABDUL REHAMAN**, solemnly declare that dissertation titled, "A STUDY OF PATIENTS PROFILE WITH ACUTE SYMPTOMATIC SEIZURES IN ANNAL GANDHI MEMORIAL GOVERNMENT HOSPITAL, TRICHY" is a bonafide work done by me at K.A.P.V. Government Medical College, during 2009-2011 under the guidance and supervision of my Unit Chief **Prof. Dr.V. RAJENDRAN M.D** Associate Professor of Medicine, Chief– Medical Unit – IV The dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University, towards the partial fulfillment of requirement for the award of M.D. Degree (Branch – I) in General Medicine.

Place: Trichy
Date : 15/12/2011

DR ABDUL REHAMAN

ACKNOWLEDGEMENT

At the outset, I thank our **Dean Prof**. **Dr. A.KARTHIKEYAN,M.D.,** for permitting me to use the facilities of Annal Gandhi Memorial Government Hospital to conduct this study

I wish to express my respect and sincere gratitude to my beloved teacher and Head of the Department of Medicine, **Prof.Dr.C.ASOK KUMAR,M.D.,** for his valuable guidance and encouragement throughout the study and also during my post graduate course. I owe my sincere thanks to him

I also owe my sincere thanks to my beloved unit chief and my guide **Prof. Dr. V.RAJENDRAN, M.D** for his guidance and support throughout the conduct of the study and also during my post graduate course.

I express my gratitude to **Registrar.Dr.N.K SENTHILNATHAN, M.D.,** Department of Medicine and K.A.P.V. Govt. Medical College, for his valuable assistance and guidance.

I owe my thanks to **Prof. Dr.M. ABDUL ALEEM, M.D., DM.,**Professor and Head of the Department of Neurology, K.A.P.V. Govt. Medical College for his help and guidance.

I am extremely thankful to **Dr.A Sethuraman, M.D., Dr.M. Ashok M.D,Dr. A.Joseph Panneerselvam DNB.,D.Diab** Assistant Professors of Medicine, K.A.P.V. Govt. Medical College, who has been the inspiration behind this study and for their unlimited encouragement, guidance and help during this study.

I owe my thanks to **Dr.V.Ravikumar, M.D.,DM.,** Associate Professor of Neurology, **Dr. S. Rajasekaran, M.D., D.M.,** for their guidance and help during this study.

I also wish to express my respect and sincere gratitude to my former Professor and HOD **Dr.S.Panneerselvam M.D.,**for his constant support and help.My sincere thanks to my former Registrar **Dr.M.Sivashankaran.MD.,**Assistant Professors **Dr.S. Manoharan.MD**.,**Dr.G.Sathish Kumar.M.D., Dr.E Arun Raj M.D.**, for their constant encouragement and help.

Last but not least, my sincere thanks to all the **patients** who co-operated for this study, without whom this study could not have been undertaken and to all my **colleagues** who helped me and shared their knowledge about this study.

Dr. Abdul Rehaman

M.D. Post Graduate

CONTENTS

ABBREVIATIONS

ANA : Anti Nuclear Antibody

APLA : Anti Phospholipid Antibody

AVM : Arteriovenous malformations

CAD : Coronary Artery Disease

CKD : Chronic Kidney Disease

CNS : Central Nervous System

CSOM : Chronic Suppurative Otitis Media

CT : Computed Tomography

CVT : Cerebral Venous Thrombosis

DM : Diabetes mellitus

dsDNA: Double standard Deoxyribo Nucleic Acid

ECG : Electrocardiogram

EEG : Electroencephalogram

EPC : Epilepsia Partialis Continua

FND: Focal Neurological Deficit

GTCS: Generalised Tonic Clonic Seizure

HIV : Human Immunodeficiency Virus

HTN : Hypertension

ICH : Intra Cerebral Hemorrhage

LFT : Liver Function Test

LS: Lateral Sinus

LVH : Left Ventricular Hypertrophy

MRI: Magnetic Resonance Imaging

MRV: Magnetic Resonance Venography

NCC : Neurocysticercosis

RHD : Rheumatic Heart Disease

SAH : Subarachnoid Hemorrhage

SCTEL : Single CT Enhancing Lesion

SS : Sagittal Sinus

SSCCCTL : Small Single Cerebral Calcific CT Lesion

SSS : Superior Sagittal Sinus

TB : Tuberculosis

NA: Not Applicable

.

INTRODUCTION

The word seizure is derived from Latin word "sacire", meaning,"to take possession of" indicating that the person having a seizure is possessed or atleast out of control[1]. The clinical symptoms in seizures could be motor, sensory, autonomic, or psychic events although in practice, when a patient presents to a health care system with a seizure it is usually a convulsive (motor) seizure, either generalized or focal[1]. Acute symptomatic seizures are defined as seizures caused or provoked by an acute medical or neurological insult. It may be single or repetitive. Type of seizure can be focal with or without generalisation. Most common is generalised tonic clonic seizure. Non convulsive seizure and status are common in patients admitted to intensive care units. Partial seizure is usually associated with structural abnormalities of brain but generalised seizure may result from cellular or structural abnormalities that have a wide spread distribution.

In developing countries most common cause of acute symptomatic seizure is CNS infections. Seizures in the elderly may be caused by stroke, systemic metabolic conditions, subdural hematoma, central nervous system infection, degenerative disorders, alcohol withdrawal or malignancy. The etiology of seizures is multi factorial in any given individual and is best thought of as an interaction between genetically determined seizure threshold, underlying predisposing Pathologies or metabolic derangements and acute precipitating factors.

In 5% of patient, trauma to head, cerebro-vascular disease and CNS Infections may manifest as acute symptomatic seizures. The occurrence of acute symptomatic seizures is associated with an additional increase in risk for epilepsy. New-onset acute symptomatic seizures can be the presenting feature of acute neurological diseases. The etiological spectrum of new- onset acute symptomatic seizures and outcome may be different in developing countries when compared to developed countries.

Approximately 60% of all epilepsies are idiopathic or cryptogenic. Cerebrovascular disease is the most commonly identified cause among elderly, while perinatal insults seem to be most common among children. Status epilepticus is one of the most common neurologic emergencies in children, adolescents and young adults. Status epilepticus may be due to acute neurologic conditions such as meningitis, encephalitis, stroke complicated febrile seizures, intractable epilepsy, degenerative diseases, intoxication, or may be the first manifestation of epilepsy.

Very few studies are there analysing the causes of acute symptomatic seizures, from south India. So it is useful to study the various conditions producing seizures in our patients and the use of investigations to find out the underlying problem. A detailed medical history, a thorough physical examination, especially of the nervous system, analysis of blood and other body fluids, electroencephalographic (EEG) recordings, magnetic resonance imaging (MRI) and/or computerized tomography (CT) scans we are able to find out the

underlying cause of acute symptomatic seizures. Accurate diagnosis of the cause of acute symptomatic seizure is very important in the treatment and prognosis as the treatment of underlying condition will abolish the seizure .Since most of the acute symptomatic seizures show a clearly identifiable cause, they usually do not recur and no need of long term antiepileptic treatment.

REVIEW OF LITERATURE

A seizure is defined as a paroxysmal event due to abnormal, excessive, hypersynchronous discharges from an aggregate of central nervous system neurons this abnormal CNS activity can have various manifestations, ranging from dramatic convulsive activity to experimental phenomena not readily discernible by an observer. About ~5–10% of the population will have at least one seizure, with the highest incidence occurring in early childhood and late adulthood.[1]

Acute symptomatic seizure was defined as seizure caused or provoked by an acute medical or neurological insult.

Acute symptomatic seizures were further grouped into two broad categories:

1) Acute symptomatic seizure caused by acute neurological insult

2) Acute symptomatic seizure caused by acute metabolic disorder[5]

Epilepsy describes a condition in which a person has recurrent seizures due to a chronic, underlying process .Epilepsy refers to a clinical phenomenon rather than a single disease entity , since there are many forms and causes of epilepsy. Using the definition of epilepsy as two or more unprovoked seizures, the incidence of epilepsy is 0.3–0.5% in different populations throughout the world, and the prevalence of epilepsy has been estimated at 5–10 persons per 1000.[1]

However, among the many causes of epilepsy there are various epilepsy syndromes in which the clinical and pathologic characteristics are distinctive

and suggest a specific underlying etiology. Seizures have been classified in several ways: according to their supposed etiology, i.e., idiopathic (primary) or symptomatic (secondary); their site of origin; their clinical form (generalized or focal); their frequency (isolated, cyclic, or repetitive, or the closely spaced sequence of status epilepticus); or their electrophysiological correlates[2]. The classification of seizure was first proposed by Gastaut in 1970 and was then refined repeatedly by the Commission on Classification and Terminology of the International League against Epilepsy (1981). This classification, based mainly on the clinical form of the seizure and its electroencephalographic (EEG) features, has been adopted worldwide and is generally referred to as the International Classification[2]. A simple classification system, another ILAE classification that was developed in 1993 for conducting epidemiological survey on epilepsy. This was named the Epidemiological Classification (EC) and was proposed for only research purposes to overcome technical problems in field studies, Now recently Classification of Seizures The International League against Epilepsy (ILAE) Commission on Classification and Terminology, 2005–2009 has provided an updated approach to classification of seizures his system is based on the clinical features of seizures and associated electroencephalographic findings

CLASSIFICATION

1. Focal Seizure

(Can be further described as having motor, sensory, autonomic, cognitive, or other feature)

2. Generalized seizures

   a. Absence

      Typical

      Atypical

   b. Tonic clonic

   c. Clonic

   d. Tonic

   e. Atonic

   f. Myoclonic

3. May be focal, generalized, or unclear

   Epileptic spasms

Another classification

Classification of seizures[28]

1. GENERALIZED SEIZURES

Tonic clonic

Absence Typical

Atypical

Absence with special features

Myoclonic absence

Eyelid myoclonia

Myoclonic Myoclonic

Myoclonic atonic

Myoclonic tonic

Clonic

Tonic

Atonic

2. FOCAL SEIZURES

* Without impairment of consciousness/responsiveness

+ With observable motor or autonomic components (simple partial seizure)

+ Involving subjective sensory or psychic phenomena only (aura)

* With impairment of consciousness/responsiveness (complex partial seizure)

* Evolving to a bilateral, convulsive seizure (replaces the term secondarily generalized seizure)

3. MAY BE FOCAL, GENERALIZED, OR UNCLEAR Epileptic spasms

Outline of the Epidemiological Classification Commission on Epidemiology and Prognosis, International League against Epilepsy[28].

2.1. Generalized seizures – when clinical symptomatology provides no indication of an anatomic localization and no clinical evidence of focal onset.

2.2 Partial seizure – when there is evidence of a clinical partial onset (by aura or focal symptoms).

2.3/2.4 Undetermined seizures – it is impossible to classify seizures owing to lack of adequate information or variable/mixed partial and generalized seizures.

Risk factors or etiology

3.1 Symptomatic seizures or epilepsies – consequence of a known cerebral dysfunction

3.1.1 Acute symptomatic – seizures are in close temporal association (within 7 days) with an acute systemic, metabolic or toxic insult and with acute CNS insult (infection, stroke, cranial trauma, intracerebral hemorrhage, acute alcohol intoxication or withdrawal).

3.1.2 Remote Symptomatic – seizures in relation to a well demonstrated antecedent condition called as remote symptomatic seizures or epilepsy (more than 7days of cerebral insult).

3.2 Unknown etiology – no clear antecedent etiology can be detected.

Basically, this classification divides seizures into two types;

FOCAL SEIZURES:

A fundamental principle is that seizures may be either focal or generalized.

Focal seizures originate within networks limited to one cerebral hemisphere. *Generalized seizures* arise within and rapidly engage networks distributed across both cerebral hemispheres. Focal seizures are usually associated with structural abnormalities of the brain. In contrast, generalized seizures may result from cellular, biochemical, or structural abnormalities that have a more widespread distribution. There are clear exceptions in both cases Instead, depending on the presence of cognitive impairment; they can be described as focal seizures with or without dyscognitive features[1]

3 Additional features are associated with Focal seizures;

1) Jacksonian march- in this abnormal motor activity begins in the very restricted area and gradually progress to involve the larger portions of the body.

2) Second, patients may experience a localized paresis (Todd's paralysis)

For minutes to many hours in the involved region following the seizure The cause of Todd's paresis is unknown, but there are two

hypotheses to its cause. The first is the depletion theory, where the motor cortex is exhausted leading to prolonged neuronal hyper polarization. The second is that there is transient inactivation of motor fibres caused by activation of NMDA receptors.

3). Third, in rare instances the seizure may continue for hours or days. This condition, termed *epilepsia partialis continua*, is often refractory to medical therapy.[1]

Focal seizures can spread to involve both cerebral hemispheres and produce a generalized seizure usually of the tonic-clonic variety. This evolution is observed frequently following focal seizures arising from a focus in the frontal lobe, but may also be associated with focal seizures occurring elsewhere in the brain.[1]

If the seizure lasts for more than 30 minutes or there is sequential seizure without recovery of consciousness it is called status epilepticus. It is a highly restricted and very persistent focal motor status epilepticus.[2]

The first solitary seizure or brief outburst of seizures may occur during the course of many medical illnesses. It indicates that the cerebral cortex has been affected by disease, either primarily or secondarily. Convulsive seizures by their nature, if repeated every few minutes, as in status epilepticus, may threaten life. Equally important, a seizure or a series of

seizures may be the manifestation of an ongoing neurologic disease that demands the employment of special diagnostic and therapeutic measures

Generalized seizures:

Generalized seizures are thought to arise at some point in the brain but immediately and rapidly engage neuronal networks in both cerebral hemispheres. It is also useful clinically and etiologically to separate epilepsies that originate as truly generalized electrical discharges in the brain from those which spread secondarily from a focus to become generalized. The primary generalized epilepsies are a group of somewhat diverse, age-dependent phenotypes that are characterized by generalized 2.5- to 4-Hz bifrontally predominant spikes or polyspike and-slow- wave discharges that arise without underlying structural abnormalities.[2] Seizures that begin locally and evolve into generalized tonic-clonic seizures, termed secondary generalized seizures, generally have no such genetic component and are usually the result of underlying brain disease, either acquired or due to congenital malformations or metabolic defects.

Epilepsy Syndromes

Epilepsy syndromes are disorders in which epilepsy is a predominant feature, and there is sufficient evidence (e.g., through clinical, EEG, radiologic, or genetic observations) to suggest a common underlying mechanism. Three important

epilepsy syndromes are listed below Juvenile Myoclonic Epilepsy Lennox-Gastaut Syndrome Mesial Temporal Lobe Epilepsy Syndrome[1]

Age distribution

Age group	Common epilepsy
Neonatal period and early infancy	Hypoxic-ischemic encephalopathy trauma, CNS infection, congenital CNS abnormalities, metabolic disorders and vitamin B6 deficiency
Late infancy and early childhood	Most common seizures are febrile seizures
Childhood	Absence seizure, myoclonic seizure, Temporal lobe epilepsy
Adolescent(10-18yrs)	Genetically transmitted, juvenile myoclonic, trauma, drugs
Early adulthood(18-25yrs)	Idiopathic, trauma, neoplasm, alcohol withdrawal
Middle age(35-60yrs)	Trauma, vascular , alcohol withdrawal
Older age(>60yrs)	Cerebrovascular disease(50%) Vascular, tumor, degenerative, trauma

.

CLINICAL FEATURES;

When a patient presents shortly after a seizure, the first priorities are attention to vital signs, respiratory and cardiovascular support, and treatment of seizures if they resume. Life-threatening conditions such as CNS infection, metabolic derangement, or drug toxicity must be recognized and managed appropriately. When the patient is not acutely ill, the evaluation will initially focus on whether there is a history of earlier seizures.

If this is the first seizure, then the emphasis will be to:

(1) Establish whether the reported episode was a seizure rather than another paroxysmal event.

(2) Determine the cause of the seizure by identifying risk factors and precipitating events. and

(3) Decide whether anticonvulsant therapy is required in addition to treatment for any underlying illness.[1]

History from a reliable attender is very important in the diagnosis of seizure.

In the diagnosis of epilepsy history is the key and the physical examination is important to identify any underlying etiology. An in-depth history is essential, for in many cases the diagnosis of a seizure is based solely on clinical grounds,the examination and laboratory studies are often normal.The general physical examination includes a search for signs of infection or systemic illness. Careful examination of the skin may reveal signs of neurocutaneous disorders, such as

tuberous sclerosis or neurofibromatosis, or chronic liver or renal disease. A finding of organomegaly may indicate a metabolic or storage disease, and limb asymmetry may provide a clue to brain injury early in development. Signs of head trauma and use of alcohol or illicit drugs should be sought. Epileptogenic factors such as prior head trauma, stroke, tumor, or infection of the nervous system should be identified Auscultation of the heart and carotid arteries may identify an abnormality that predisposes to Cerebrovascular disease.[1,2]

All patients require a complete neurologic examination, with particular emphasis on eliciting signs of cerebral hemispheric disease. Careful assessment of mental status (including memory, language function, and abstract thinking) may suggest lesions in the anterior frontal, parietal, or temporal lobes[1,2].

Testing of visual fields will help to screen for lesions in the optic pathways and occipital lobes. Screening tests of motor function such as pronator drift, deep tendon reflexes, gait, and coordination may suggest lesions in motor (frontal) cortex, and cortical sensory testing (e.g., double simultaneous stimulation) may detect lesions in the parietal cortex.

Investigations:

Biochemical investigations are very much helpful in case of acute symptomatic seizures Secondary to alteration in biochemical parameters. Among these , most important are imbalance in electrolytes , glucose, calcium, or magnesium levels. In suspicious patients, estimation of toxins can be done in blood as well as urine sample.Systemic acidosis is a common result of convulsive seizures, almost all

generalized convulsions produce a rise in serum creatine kinase activity that persists for hours, a finding that could be used to greater advantage in emergency departments to assist in distinguishing seizures from fainting. Concentrations of serum prolactin, like those of other hypothalamic hormones, rise for 10 to 20 min after all types of generalized seizures, including complex partial seizures, but not in absence or myoclonic types. An elevation may help differentiate a hysterical seizure from a genuine one. A lumbar puncture is indicated if there is any suspicion of meningitis or encephalitis, and it is mandatory in all patients infected with HIV, even in the absence of symptoms or signs suggesting infection. A significant pleocytosis after a seizure should always be construed as a sign of inflammatory or infectious disease[2].

Electroencephaogram

The EEG is undoubtedly the most sensitive, indeed indispensable, tool for the diagnosis of epilepsy; but like other ancillary tests, it must be used in conjunction with clinical data. In all patient with suspected epilepsy, it is valuable to do electroencephalogram which shows the presence of electrographic activity during clinically evident event. Since seizures are typically infrequent and unpredictable, it is often not possible to obtain the EEG during a clinical event. Continuous monitoring for prolonged periods in video-EEG telemetry units for hospitalized patients or the use of portable equipment to record the EEG continuously on cassettes for 24 h in ambulatory patients has made it easier to capture the electrophysiological accompaniments of clinical events. In particular,

video-EEG telemetry is now a routine approach for the accurate diagnosis of epilepsy in patients with poorly characterized events or seizures that are difficult to control However absence of electrographic activity does not rule out seizure because simple or complex seizures may originate from a region of the cortex which is not within the range of scalp electrodes . The EEG may also be helpful in the interictal period, shows epileptiform activity The presence of epileptiform activity is not specific for epilepsy, but it has a much greater prevalence in patients with epilepsy than in normal individuals. A single EEG obtained during the interictal state is abnormal to some degree in 30 to 50 percent of epileptic patients; this figure rises to 60 to 70 percent if patients are subjected to three or more studies utilizing standard activating measures (hyperventilation, photic stimulation, and sleep).[2]Focal interictal epileptiform discharges would support the diagnosis of a partial seizure disorder such as temporal lobe epilepsy or frontal lobe seizures, depending on the location of the discharges. In general, a normal EEG implies a better prognosis, whereas an abnormal background or profuse epileptiform activity suggests a poor outlook[16, 1,2,8]

Magneto encephalography (MEG) measures small magnetic fields that are generated by the seizure activity. It is a non-invasive mode of investigation which localizes the focus of seizure activity by measuring the magnetic field and analyzing its location on a anatomic image of brain.[9]

BRAIN IMAGING

Almost all patients with new-onset seizures should have a brain imaging study to determine whether there is an underlying structural abnormality that is responsible.[10]

MRI is the most important diagnostic tool for the detection of structural abnormalities underlying epilepsy. MRI has been shown to be superior to CT for the detection of cerebral lesions associated with epilepsy.[2] In some cases MRI will identify lesions such as tumors, vascular malformations, Medial temporal sclerosis, glial scars, porencephaly, heterotopias, and other disorders of neuronal migration or other pathologies that need immediate therapy can be clearly visualized.[2] The use of newer MRI methods, such as fluid-attenuated inversion recovery(FLAIR), has increased the sensitivity for detection of abnormalities of cortical architecture, including hippocampal atrophy associated with mesial temporal sclerosis, as well as abnormalities of cortical neuronal migration[11,12]. Volumetric MR Imaging gives quantitative evaluation of hippocampal volume has been found to marginally increase the sensitivity over visual analysis in detection of hippocampal sclerosis. T2 Relaxometry is used to quantify the T2 signal in the hippocampus, in mesial temporal sclerosis the relaxation time has proven to be lengthened by 10 milliseconds. MR Spectroscopy has been widely used in providing insight into the metabolic alterations in epilepsy [1,2].

In the patient with a suspected CNS infection or mass lesion, CT scanning should be performed .Functional imaging procedures such as positron emission tomography (PET) and single photon emission computed tomography (SPECT) are also used to evaluate certain patients with medically refractory seizures.[6]

DEMOGRAPHY

Epidemiology of acute symptomatic seizure varies in different age groups and in different countries .one study[8] showed that seizure occurred in close temporal association with an acute central nervous system (CNS) insult in 53% of patients. Cerebrovascular diseases were the risk factors in 48% of patients with remote symptomatic epilepsy. Infections of CNS including single CT enhancing lesion (SCTEL) accounted for 77% of patients with acute symptomatic epilepsy. Neurocysticercosis, SCTEL and small single cerebral calcific CT lesion (SSCCCTL) together accounted for 40% of etiological factors and neurotuberculosis for 10%. Infections of the central nervous system and SCTEL together were the putative risk factors in 52% of patients aged ≤40 years. The type of seizure was either simple partial or complex partial with or without secondary generalization in 76% of patients.

Another study showed that[12] the etiological risk factors were central nervous system infections in 32% patients, metabolic disorders in 32%, cerebrovascular diseases in 21% and other causes in 15%. In CNS infections, meningoencephalitis is seen 43% and parenchymal granuloma in 57% of

patients. In that,degenerative phase solitary cystic granuloma in 75%and tuberculoma in 25% of patients.

According to study conducted by Sander et al[26] showed

Febrile seizure	20%	AGE	%
Definite epileptic seizure	52%	<15yrs	25%
Possible epilepsy	21%	>60yrs	24%

In the definite epilepsy group the proportions of males and females were similar

The definite seizures were classified as

Cryptogenic	62%
Remote symptomatic	21%
Acute symptomatic	15%

The etiology of epilepsy was vascular disease in 15% (12-18%) and tumor in 6% (4-8%). Among older subjects the proportion with an identifiable cause was much higher: 49% (41-58%) were due to vascular disease and 11% (6-16%) to tumor[26]

Developed countries most common cause is cerebrovascular disease. Cerebrovascular disease may account for ~50% of new cases of epilepsy in patients older than 65. Acute seizures i.e., occurring at the time of the stroke are seen more often with embolic rather than hemorrhagic or thrombotic stroke. Chronic seizures typically appear months to years after the initial event and are

associated with all forms of stroke. The reported incidence of post-stroke seizures varies widely between epidemiological studies, ranging from 2% to 33% for early seizures and 3% to 67% for late seizures.[3]

According to Joseph et al, Cerebrovascular disease is the most commonly identified cause of acquired epilepsy. Post-stroke seizures account for 11% of all epilepsy, 22% of all cases of status epilepticus and 55% of newly diagnosed seizures amongst older patients.

Maurizio et al study showed the risk of first seizure was increased in

- Cortical involvement
- Multiple CT-scan lesions
- Supratentorial lesions
- Prior lesions on CT-scan
- Family history of seizures
- Use of Epileptogenic drugs
- Large lesions
- Hemorrhagic lesions
- Cortical atrophy.

Head trauma is a common cause of epilepsy in adolescents and adults. A mild head injury defined as a concussion with amnesia or loss of consciousness of <30 min . A patient with a penetrating head wound, depressed skull fracture, intracranial hemorrhage, or prolonged post-traumatic coma or amnesia has a 40-

50% risk of developing epilepsy, while a patient with a closed head injury and cerebral contusion has a 5–25% risk. Recurrent seizures usually develop within 1 year after head trauma, although intervals of 10 years are well known.

Patients with CVT present with varying combinations of headache, seizures, aphasia, behavioral abnormalities, altered sensorium and focal deficits. Seizures may be focal, multi focal or generalized. The presentation is acute in obstetric and infectious CVT while a slowly progressive disease is more common in inflammatory and idiopathic cases. Thrombosis involves the dural sinuses as well as cortical veins producing cerebral infarction and neurological deficit .

CNS infections:

Infections are important cause of seizures in developing countries. Viral, bacterial, fungal and parasitic infections can result in seizures.

Bacterial infection:

Tuberculomas are granulomatous mass lesions composed of a central zone of caseation surrounded by a collagenous tissue capsule arising in the brain parenchyma or the spinal cord. Brain tuberculomas make up 5 to 8 per cent of intracranial masses in person in developing countries[17].Before effective chemotherapy was available for tuberculosis. tuberculoma made up 20 per cent of intracranial lesions in one large series [19]

The incidence of Neurotubeculosis in the United States is less than 0.5 per cent[18]The commonest presenting symptoms[20] were headache (100%), partial or generalized convulsions (68.7%) and hemiparesis with or without

hemisensory symptoms (56.2%).The role of neuro-imaging by CT or MRI scan in the diagnosis of Tuberculoma is well-established . MRI is superior to CT in visualizing the morphological details of Tuberculoma, and particularly the tiny brain stem lesions. Improvements in diagnostic efficacy have been made possible by utilizing MR diffusion weighted imaging, spectroscopy and minimally invasive CT-guided biopsy. The few studies available from developing countries on the frequency of radiological clearance have shown complete resolution of the intracranial lesions in 80–100% of patients on shortcourse (6–12 months) chemotherapy. A recent study from India, including only histopathologically verified cases, revealed a lower rate of 54% complete resolution by 24 months of treatment.[20] According to Jayasree et al[12] 32% of cases of seizures were due to CNS infections, of this 4.5% contributed by tuberculoma .

Viral infection:

The virus causing aseptic meningitis includes entero-virus(more common in developing countries because of faecal-oral transmission), mumps, arena viruses, herpes simplex type-2, varicella zoster and HIV.[2] Herpes simplex encephalitis has no pathognomonic clinical presentation but presents as focal encephalitis with malaise; focal seizures that may become generalized Herpes simplex encephalitis produces dramatic electroencephalographic (EEG) focal ,temporal or lateralized polymorphic delta activity as the earliest Changes. CT and MRI reveal medial temporal involvement.[14]

Japanese encephalitis is caused by mosquito-borne Japanese encephalitis virus. In Japanese encephalitis convulsions may occur as part of severe encephalitis and the mortality rates are high (20% to 40%). MRI shows thalamic and basal ganglia involvement.[3,14]

With the upsurge in HIV infection this may be an important cause for acute symptomatic seizures. Seizures may rarely be the presenting manifestation of HIV infection.Opportunistic infections such as toxoplasmosis, tuberculosis, progressive multifocal leucoencephalopathy (PML), cryptococcal meningitis and polymicrobial infections, metabolic and electrolyte disturbances, and drugs are common causes of new-onset seizures in HIV. In the absence of any cause, primary HIV infection may be considered responsible for seizures .

Parasitic infection:

Malaria is caused by plasmodium species and the most common fatal parasitic disease. Approximately 2% of all patients with malaria have cerebral involvement and nearly 80% of patients who ultimately die have cerebral involvement. Cerebral malaria is fatal in 20% to 50% of affected patients In a recent report from Nigeria where malaria is endemic, cerebral malaria is responsible for one third of seizures with fever in childhood.[16]

Neurocysticercosis is a disease caused by the infection with the larval stage of the intestinal cystode. Taenia solium that occurs when human or porcine become intermediate hosts The parasite has marked tendency to infect muscle and the central nervous system where it produces a pleomorphic clinical disorder

neurocysticercosis In many developing countries neurocysticercosis is the most common parasitic disease of the central nervous system and accounts for 10% of all acute neurological diseases. There are wide variations of clinical manifestations of neurocysticercosis. These are consequence of inflammation around a cyst, space occupation and impedance to the flow of cerebrospinal fluid. Epilepsy is the most common manifestation of Neurocysticercosis, occurring in two-third of affected patients.[4,5]

Acute symptomatic seizures occur during the focal encephalitic illness caused by degenerating parasite but chronic epileptogenic focus that causes late epilepsy develops due to healing by peri-lesional gliosis and chronic calcified lesion . Seizures in Neurocysticercosis are generalized convulsive or simple partial with focal motor activity.

. Del Brutto et al[15]. studied clinical characteristics of 203 patients with epilepsy and Neurocysticercosis and found generalized convulsive seizures in 38% and complex partial seizures in 2%. Medina et al. in their series of 50 patients with epilepsy due to neurocysticercosis[26] found that 72% had partial seizures Neuro-imaging is essential to the diagnosis of neurocysticercosis. Brain MRI is superior for showing intraventricular or subarachanoid cyst and for showing inflammation around a cyst whereas CT is better for showing the calcification of inactive lesions. However, recent MRI studies with gradient echo and reversed gradient echo phase image have shown scolex visible within the calcified lesion as visible on CT scan. These entrapped antigens have been shown to be responsible for

intermittent immuno-allergic response, peri- lesional edema and seizure recurrence. There may be single or multiple cysts in different pathological stage[15]. Carpio has proposed a classification system that corresponds to the viability of the parasite: active, transitional and inactive. Both CT and MRI can show the presence of the eccentric mural nodule (the invaginated scolex), an appearance when multiple is pathognomonic of neurocysticercosis (starry night appearance). Magnetization transfer spin echo sequence of MRI and calculation of magnetization transfer ratios used to differentiate neurocysticercosis from tuberculoma[16]

Cerebral toxoplasmosis is caused by protozoan Toxoplasma gondi. It produces nonspecific signs and symptoms of intracranial mass lesion and seizures in immunocompromized patients. In the developing world toxoplasmosis may occur without HIV infection as well and linear beaded appearance on MRI may be diagnostic .Focal neurological deficits occurred in (69%) and seizure in (29%).

Metabolic disorder

Various metabolic disorder causing seizures are electrolyte imbalance, hypo- or hyperglycemia, renal failure, and hepatic failure, hypernatremic hyperosmolar state, thyrotoxic storm, porphyria , hypomagnesemia,and hypocalcemia. Rapidly evolving electrolyte abnormalities are more likely to cause seizures than those occurring gradually. Similarly endocrine disorders, hematologic disorders, vasculitis, and many other systemic diseases may cause seizures over a broad age range. A wide variety of medications and abused substances are known to

precipitate seizures ex(Imepenam penicillin congeners,cefapime,TCA, bupropion, lithium lignocaine tramadol).[1,2]

In case of Diabetic nonketotic hyperosmolar patient usually presents with clouded sensorium, partial motor seizures, transient hemiplegia, chorea, hemiballismus and hemichorea – hemiballismus.[24]

Renal failure patient may manifest a variety of neurologic disorders. Patients with chronic renal failure who have not yet received dialysis therapy may develop a symptom complex progressing from mild sensorial clouding to delirium and coma, with tremor, asterixis, multifocal myoclonus, and seizures. These central nervous system disorders are referred to as uremic encephalopathy. As uremia progresses, it has been proposed that the accumulation of guanidino compounds results in activation of excitatory N-methyl-D-aspartate (NMDA) receptors and inhibition of inhibitory GABA receptors, which may cause myoclonus and seizures. Seizures in patients with uremia may be due to:

- Hypertension
- Electrolyte imbalance
- Aluminum toxicity
- Drug toxicity
- Infection
- Elevation of intracellular sodium

- Accumulation of toxic metabolites
- Inhibition of GABA responses

The dialysis treatment of end-stage renal disease has itself been associated with the emergence of two distinct new disorders of the central nervous system; dialysis dysequilibrium and dialysis dementia . A study[13] of nine patients, aged 23 to 67 years, showed a remarkable sequence of EEG findings in progressive uremic encephalopathy. The initial characteristics suggested a disorder of subcortical gray matter, followed by involvement of cortical gray matter and finally white matter. Seizures indicated a grave prognosis.[13]

When seizures occur in the context of renal insufficiency, it is necessary to rule out a number of complications other than uremia:electrolyte imbalance (water intoxication,hypocalcemia,hyponatremia,hypomagnesemia)aluminum encephalopathy ,drug intoxication, hypertensive encephalopathy,intracranial hemorrhage ,subdural hematoma and Wernicke's encephalopathy. seizures due to hyponatremia was investigated in five patients with epilepsy and polydipsia–hyponatremia. They experienced marked increases in the frequency of their complex partial seizures with a decrease in the serum sodium level to 118–127 mEq/L . In all cases the serum sodium level returned to normal through restriction of fluids, and the clinical seizures improved.

In case of organophosphorus poisoning, seizure incidence is 22-25% in children and 2-3% in adult it causes mainly flaccid paralysis which can mask

convulsion. EEG is more reliable indicator of seizures [36.]. Seizures can rapidly progress to status epilepticus, contributing to mortality and, in survivors, to neuronal damage and neurological impairment. Anticonvulsant drugs can significantly reduce the lethal and toxic effects of these compounds A benzodiazepine, usually diazepam, is the treatment currently indicated for control of seizures. Neuropathology caused seizures is most likely associated with glutaminergic excitotoxicity. Future prospects for improved treatments include new benzodiazepines, glutamate receptor antagonists, antimuscarinics with additional antiglutamatergic activity and adenosine receptor antagonists .The illegal mixing of organophosphates and pyrethroids in marketed agriculture insecticides in developing countries cause combination of miosis, bradycardia, tachypnea, and unconsciousness and seizures. The occurrence of pupillary dilation after a small-dose infusion of atropine (0.08 to 0.2 mg/kg in 1–3 h) and seizures raise the possibility of pyrethroid poisoning.

Alcohol Withdrawal siezure is seen when an individual reduces or stops alcohol consumption after prolonged periods of excessive alcohol intake. In about 23-33% of patients with significant alcohol withdrawal have alcohol withdrawal seizures("rum fits").Seizures are usually brief, generalized, tonic-clonic in nature, and without an aura. Partial seizures are not uncommon. The incidence peaks 24 hours after the most recent alcohol ingestion. Most seizures typically terminate spontaneously or are easily controlled with benzodiazepines. There is little evidence that anticonvulsants such as phenytoin or gabapentin are more effective

in drug-withdrawal seizures, and the risk of seizures has usually passed by the time effective drug levels are reached. Status epilepticus may occur in 3% of alcohol withdrawal seizures and should prompt an investigation for other causes, as people with alcoholism are prone to head injuries, chronic idiopathic epilepsy, and meningitis.[34]

CNS tumors are more commonly associated with partial seizure. In 75% of cases, Oligodendroglial brain tumours presented with symptoms related to seizures. The underlying pathophysiologic mechanisms of tumor-associated seizures are of altered peri tumoral amino acids, regional metabolism, pH, neuronal or glial enzyme and protein expression, as well as immunologic activity. Studies are needed to elucidate more clearly the pathophysiologic mechanisms of tumor-related seizures and to identify and develop the optimal AEDs.[35]

TREATMENT

The treatment of seizures of all types can be classified into

- Removal of causative and precipitating factors
- Use of antiepileptic drugs
- suppression of recurrent seizures by prophylactic therapy
- Surgical excision of epileptic foci
- addressing a variety of psychological and social issues
- Regulation of physical and mental activity[1]

Treatment of Underlying Conditions

If the sole cause of a seizure is a metabolic disturbance, then treatment is aimed at reversing the metabolic problem and preventing its recurrence. Therapy with antiepileptic drugs is usually unnecessary unless the metabolic disorder cannot be corrected promptly and the patient is at risk of having further seizures. If the apparent cause of a seizure was a medication (e.g., theophylline) or illicit drug use (e.g., cocaine), then appropriate therapy is avoidance of the drug; there is usually no need for antiepileptic medications unless subsequent seizures occur in the absence of these precipitants.[1]

Initiating therapy in a patient with a single seizure is controversial . Patients with lesions like Neurocysticercosis ,Tuberculoma ,vascular malformation ,brain

abscess or single enhancing lesion brain tumor need AED until resolution of lesion and maintain on antiepileptic medication for at least 1 year and an attempt is made to withdraw medications only if the patient has been completely seizure-free[1]. If seizures are refractory to medication, the patient may benefit from surgical removal of the epileptic brain region.[1]

When to Initiate Antiepileptic Drug Therapy

Antiepileptic drug therapy should be started in any patient with recurrent seizures of unknown etiology or a known cause that cannot be reversed. Generally accepted risk factors associated with recurrent seizures include the following:

(1) an abnormal neurologic examination (2) seizures presenting as status epilepticus (3) postictal Todd's paralysis (4) a strong family history of seizures or (5) an abnormal EEG. Most patients with one or more of these risk factors should be treated.[1]

CHOICE OF ANTIEPILEPTIC DRUGS

Worldwide, older medications such as phenytoin, valproic acid, carbamazepine, phenobarbital, and ethosuximide are generally used as first-line therapy for most seizure disorders since; they are as effective as recently marketed drugs and significantly less expensive. Most of the new drugs that have become available in the past decade are used as add-on or alternative therapy, although some are now being used as first-line monotherapy.

TYPE OF SEIZURE	FIRST LINE	ALTERNATIVES
Generalised tonic clonic seizure	Valproic acid,lamotrigine, Topiramate	Zonisamide,phenytoin, Carbamazepine,oxcarbamazepine,phenobarbitol, Primidone,felbamate
Focal	Lamotrigine, carbamazepine,oxcarbamazepine, phenytoin,levetiracetam	Topiramate,zonisamide, valproicacid,tiagabine,gabapentin,lacosamide phenobarbitol, Primidone,felbamate
Typical absence	Valproic acid,ethosuximide	Lamotrigine,clonazepam
Atypical absence,myoclonic, Atonic	Valproic acid,lamotrigine, Topiramate	Clonazepam, felbamate

Single seizure after stroke may not be treated and if there is high risk for reccurence AED can be given . Gabapentin and lamotrigine are the first line drugs for post stroke seizure. Patients with brain tumors have to be started with AED . If seizures are refractory to medication, the patient may benefit from surgical removal of the epileptic brain region .Patients with uremic seizures safer AED‘s are lamotrigine, valproate and phenytoin. Empirical therapy of Tuberculous meningitis is often initiated on the basis of a high index of suspicion without adequate laboratory support. Initial therapy is a combination of isoniazid (300 mg/d), rifampin (10 mg/kg per day), pyrazinamide (30 mg/kg per day in divided doses), ethambutol (15–25 mg/kg per day in divided doses) and pyridoxine (50 mg/d). When the antimicrobial sensitivity of the *M. tuberculosis* isolate is known, ethambutol can be discontinued. If the clinical response is good, pyrazinamide can be discontinued after 8 weeks and isoniazid and rifampin continued alone for the next 6–12 months. A 6-month course of therapy is acceptable, but therapy should be prolonged for 9–12 months in patients who have an inadequate resolution of symptoms of meningitis or who have positive mycobacterial cultures of CSF during the course of therapy. Dexamethasone therapy is recommended for HIV-negative patients with tuberculous meningitis. The dose is 12–16 mg per day for 3 weeks, then tapered over 3 weeks.

For brain abscess high dose parentral antibiotics and surgical drainage are advised.

Neurocysticercosis is treated with Albendazole (15 mg/kg per day for 8–28 days) or Praziquantel (50–100 mg/kg daily in three divided doses for 15–30 days). Longer courses are often needed in patients with multiple subarachnoid cysticerci If parenchymal lesions resolve without development of calcifications and patients remain free of seizures, antiepileptic therapy can usually be discontinued after 1–2 years. For patients with CVT anticoagulants initially intravenous followed by oral and antibiotics if it is septic thromboplebitis are employed. For patients with CVT, use of AED is recommended for one year.[1]

Patients with status epilepticus should be treated promptly as the condition is associated with high mortality and cardiorespiratory dysfunction, hyperthermia, and metabolic derangements can develop as a consequence of prolonged seizures, and these can lead to irreversible neuronal injury . Quick assessment of cardiorespiratory function and airway and insert large-bore intravenous line and draw blood for glucose, blood urea nitrogen, electrolytes, and a metabolic and drug screen. A normal saline infusion is begun and a bolus of glucose is given (with thiamine if malnutrition and alcoholism are factors). Diazepam is given intravenously at a rate of about 2 mg/min until the seizures stop or a total of 20 mg has been given Or lorazepam, 0.1 mg/kg given by intravenous push at a rate not to exceed 2 mg/min . A loading dose 20 mg/kg of phenytoin is administered by iv at a rate of less than 50 mg/min or fosphenytoin at 150 mg/min.If the seizure is

not controlled repeat phenytoin 7-10mg/kg. Consider sodium valproate 25mg/kg or phenobarbitone 20mg/kg infusion if the seizure still continues. Admit in ICU and give iv anaesthesia with midazolam or propofol as next step .Once the seizure is controlled continue maintanance AED.[1]

AIMS AND OBJECTIVES

The aims of the study were as follows

- To Study the etiological profile of new onset acute symptomatic seizures in patients >12 years of age.
- To study the pattern of seizures and associated features.
- To study the usefulness of various laboratory investigations in the diagnosis of acute symptomatic seizures .
- To study the incidence of potentially curable causes of seizures

MATERIALS AND METHODS

THE STUDY GROUP

The study was conducted on patients admitted in medical wards and Intensive medical care unit of Annal Gandhi Memorial Government Hospital Trichy. Approval from the hospital ethical committee was obtained.

STUDY DESIGN

The study was a cross sectional study conducted for a period of 17 months

Between June 2010- Oct 2011.

INCLUSION CRITERIA

1. Age more than 12 years.

2. Patients admitted with first episode of seizure.

3. In patients who developed first seizure in this hospital.

EXCLUSION CRITERIA

1. Idiopathic seizures

2. Patients with past history of seizure

3. Alcohol and Toxin induced seizures

METHODS

Consecutive patients with new-onset acute symptomatic seizure as the first presenting event with acute illness admitted to intensive medical care unit and medical wards of Annal Gandhi memorial government Hospital , Trichy were studied . A total number of 141 patients were studied out of which 100 were included in the study as per inclusion and exclusion criteria. All the details were noted in a specially prepared proforma a copy of which is annexed. All patients were from low socioeconomical status. A detailed history was elicited from the relatives about the type of seizure duration ,associated symptoms like fever ,headache, vomiting ,weakness or loss of consciousness. Past history of medical illness or neurological illness was elicited. Detailed examination especially

neurological, was done to find out any etiological factors focal neurological deficits or complications. Fundus examination was done to look for papilledema, or retinopathy .Blood pressure was checked and categorised to different stages according to JNC VII classification.

Baseline investigations done to find out metabolic problems, renal function, liver function and electrolyte imbalances. ECG was done for all patients to find out any cardoivascular abnormality .CSF examination done for indicated patients. Neuroimaging was done for all patients with seizures as an emergency mainly CT brain .MRI brain was done only if the CT is inconclusive or diagnosis is doubtful or if there was need for imaging of sinuses and venous system . CT or MR angiogram was done for patients with CVT , AVM or other vascular abnormalities .Special MRI sequences like FLAIR ,DWI, MR spectroscopy were done to differentiate between different lesions .Patients were treated according to underlying conditions and type of seizure . Statistical Tools The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2008). Using this software range, frequencies, percentages, means, standard deviations were calculated.

RESULTS AND ANALYSIS

Epidemiology

Majority of the patients were from in and around Trichy.

Age of patients varied from 13 to 91yrs Majority of patients were from low socioeconomic class.

Table 1: Sex distribution

SEX	NUMBER	PERCENTAGE
MALE	63	63
FEMALE	37	37
TOTAL	100	100

Out of 100 cases 63 were male 37 female

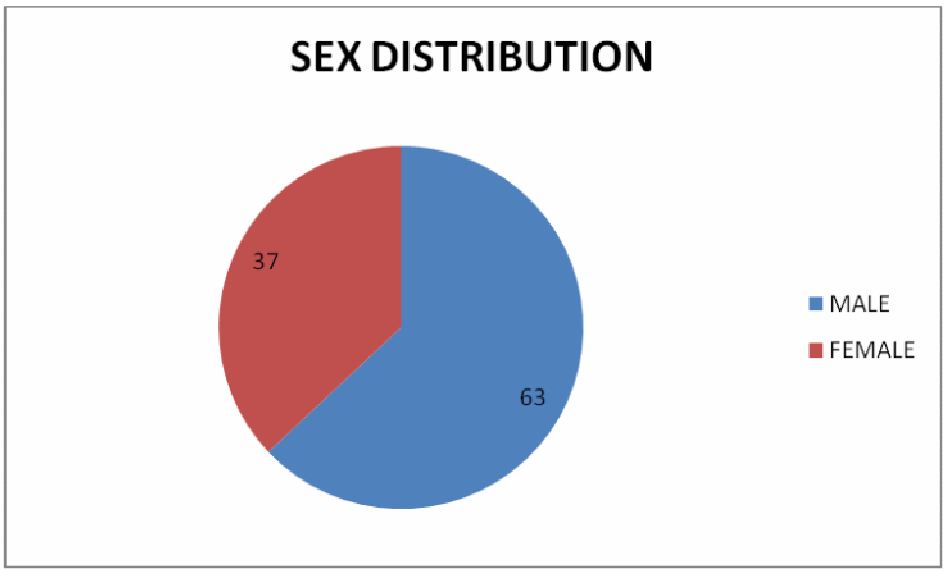
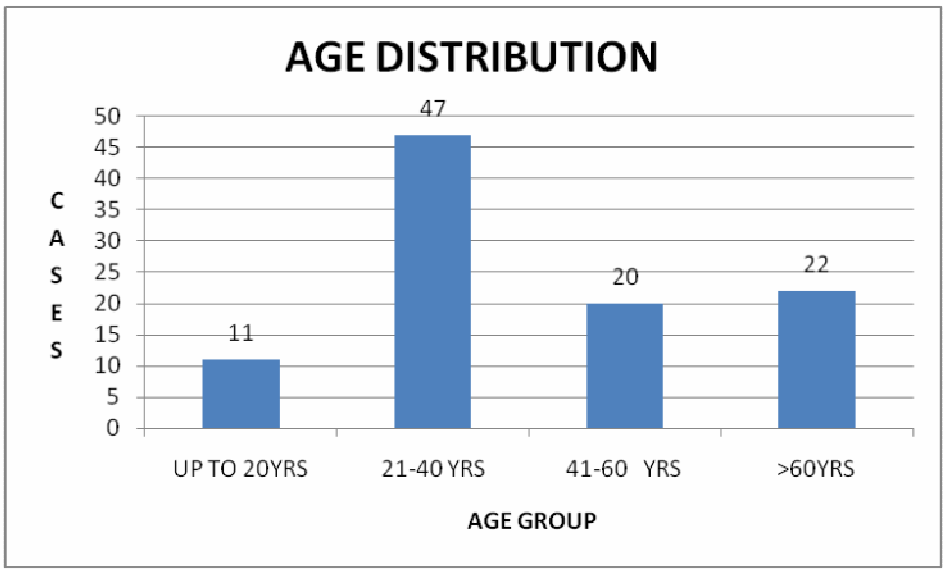


Table 2: Age distribution of patients

AGE GROUP	CASES	PERCENTAGE
UP TO 20YRS	11	11
21-40 YRS	47	47
41-60 YRS	20	20
>60YRS	22	22
TOTAL	100	100



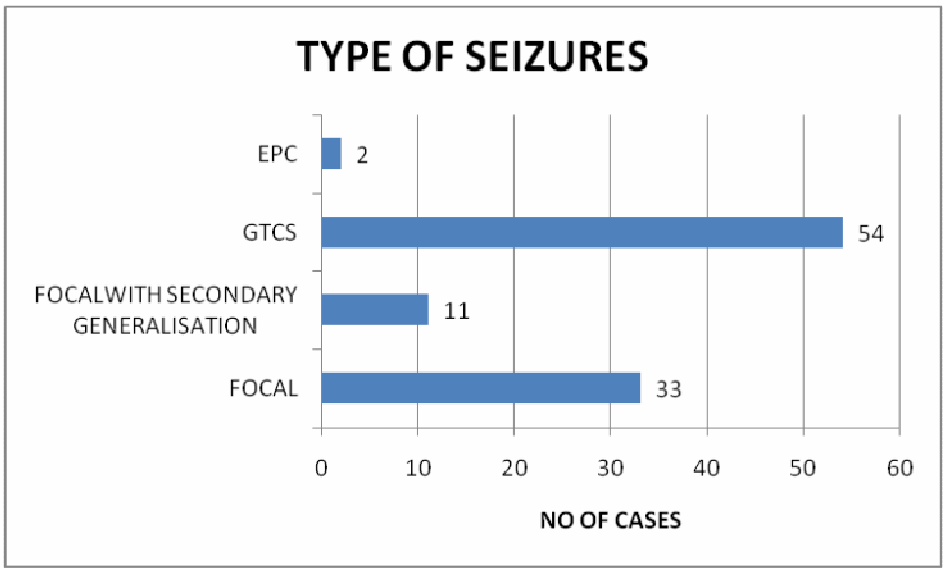
Range13-91 years

Mean 39.57 years

S.D. 16.6 years

Table 3: Type of Seizure

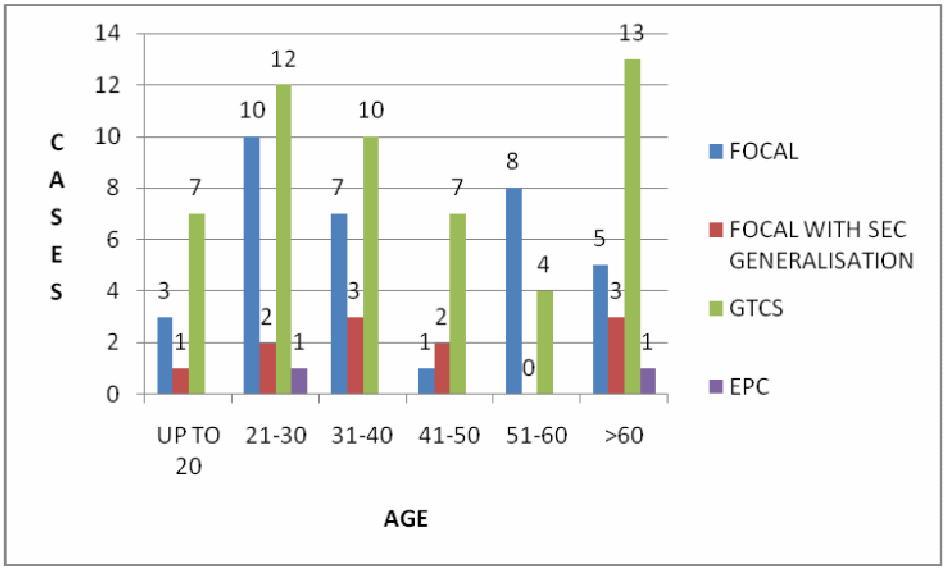
TYPE OF SEIZURES	NUMBER	PERCENTAGE
FOCAL	33	33
FOCALWITH SECONDARY GENERALISATION	11	11
GTCS	54	54
EPC	2	2
TOTAL	100	100



Out of 100 cases 33 patients presented with focal seizures, 11 with focal seizure with secondary generalization and 54 with GTCS .Only 2 patients were presented with epilepsia partialis continua. Out of these 100 cases 27 had Status epilepticus. Most common presentation was generalised tonic clonic seizures

Table 4: Type of Seizure and Age

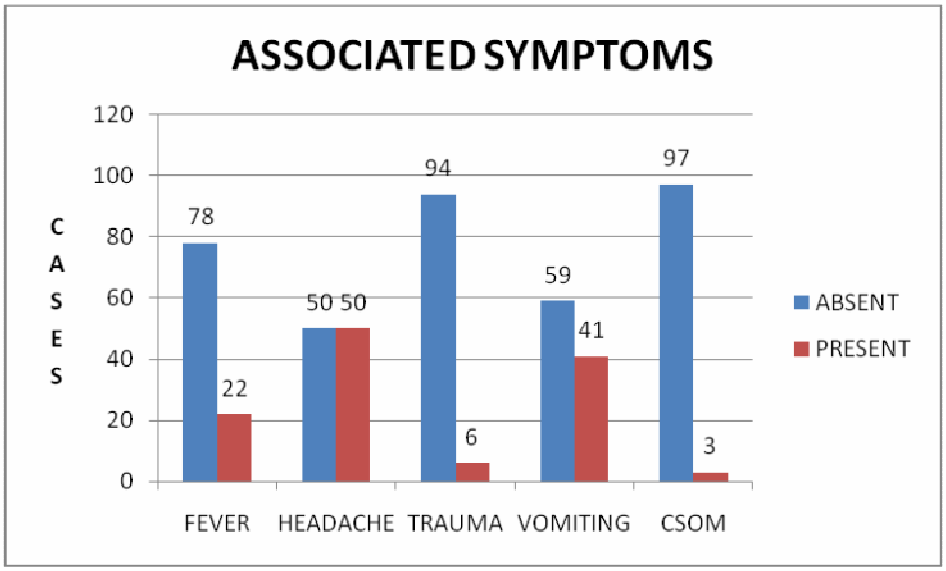
AGE GROUP	FOCAL	FOCAL WITH SEC GENERALISATION	GTCS	EPC
UP TO 20	3	1	7	-
21-30	10	2	12	1
31-40	7	3	10	-
41-50	1	2	7	-
51-60	8	-	4	-
>60	5	3	13	1



In all age groups the most common type of seizure was GTCS. Focal seizure was common in middle age. Patients presented with EPC was older adults or elderly, GTCS was most common in young patients

Table 5: Associated Symptoms

SYMPTOMS	PRESENT	ABSENT
FEVER	22	78
HEADACHE	50	50
TRAUMA	6	94
VOMITING	41	59
CSOM	3	97



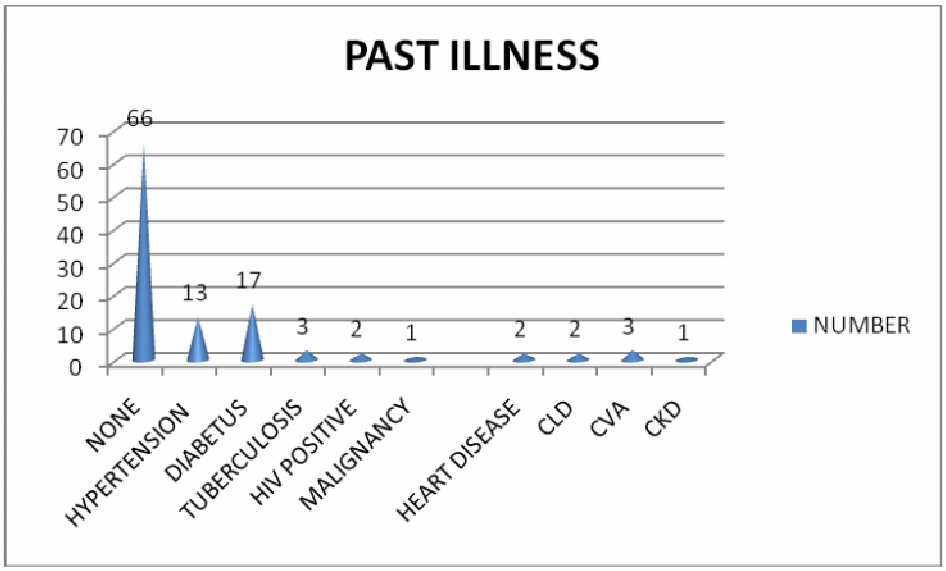
Patients with acute symptomatic seizures due to infections and CVT had headache, vomiting and fever. Only 22 patients had fever at the time of seizures. Patients with meningitis ,brain abscess ,encephalitis were presented with fever .

Headache and vomiting was there for around 50 patients. Most of these patients had mass lesions like tumor, Neurocysticercosis, Tuberculoma or abscess and CVT.

History of CSOM was there for 3 patients with brain abscess and septic thrombosis of veins.

Table 6: Past Illness

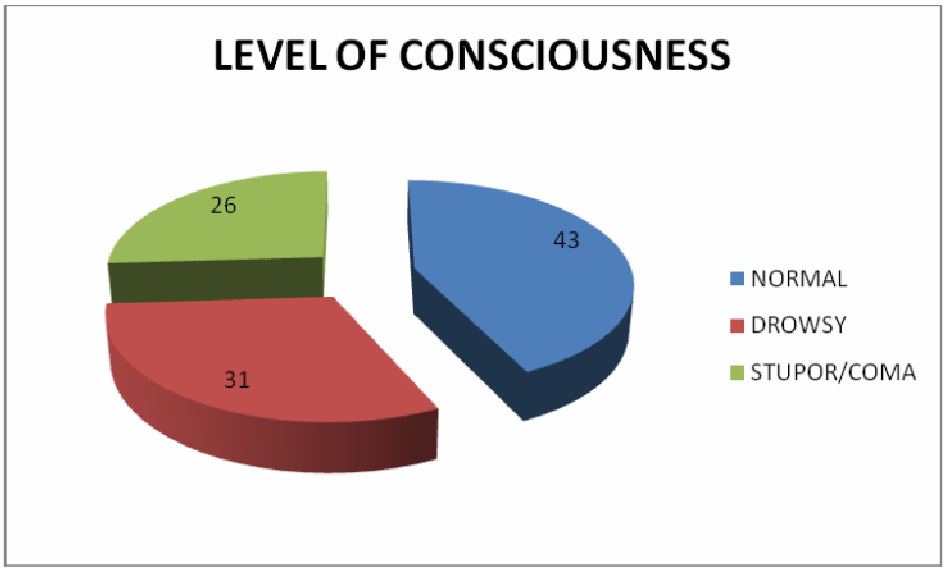
PAST ILLNESS	NUMBER	PERCENTAGE
NONE	66	66
HYPERTENSION	13	13
DIABETES	17	17
TUBERCULOSIS	3	3
HIV POSITIVE	2	2
MALIGNANCY	1	1
HEART DISEASE	2	2
CLD	2	2
CVA	3	3
CKD	1	1



Out of 100 patients 17 patients had history of diabetes and 13 patients had history of hypertension. History of treatment for malignancy of lung was present in 1 patient, 2 patients presented with GTCS were HIV positive and three patient had history of tuberculosis, fourteen cases had more than one past illness.

Table 7: LEVEL OF CONSCIOUSNESS

LEVEL OF CONSCIOUSNESS	NUMBER OF CASES	PERCENTAGE
NORMAL	43	43
DROWSY	31	31
STUPOR/COMA	26	26
TOTAL	100	100



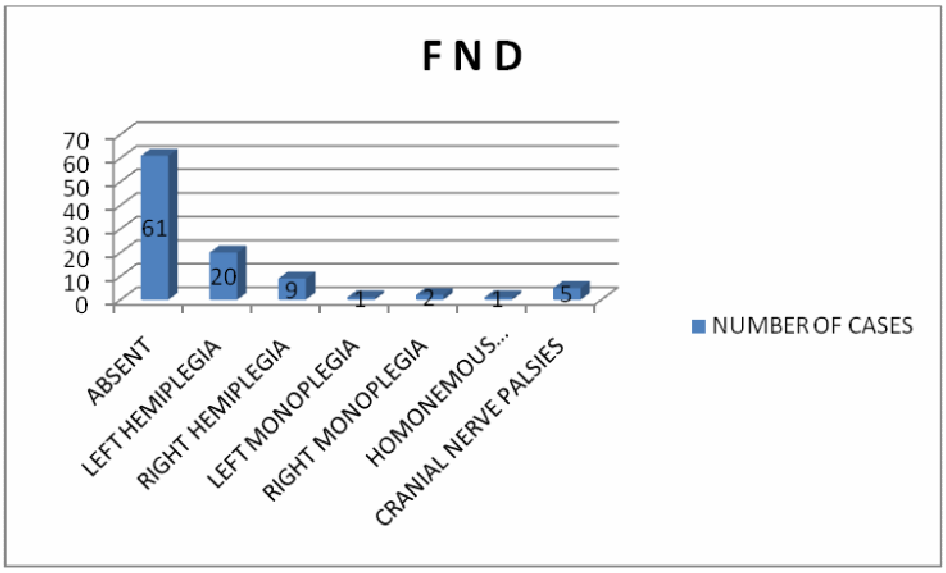
Normal level of consciousness was present only for 43 patients. All the other patients had some change in level of consciousness. Out of 100 patients 31 were drowsy and 26 patients were in stupor or coma. Ophthalmoscopic examination

was normal in 81 patients and there was papilledema for 9 cases and retinopathy for 10 cases. Blood pressure was high in 31 patients of these 21 patients were in Pre- hypertension stage, 5 in stage 1 hypertension and 15 were in stage 2 hypertension.

.

Table 8 Focal Neurological Deficit

FOCAL NEUROLOGIAL DEFICIT	NUMBER OF CASES	PERCENTAGE
ABSENT	61	61
LEFT HEMIPLEGIA	20	20
RIGHT HEMIPLEGIA	9	9
LEFT MONOPLEGIA	1	1
RIGHT MONOPLEGIA	2	2
HOMONEMOUS HEMIANOPIA	1	1
CRANIAL NERVE PALSIES	5	5

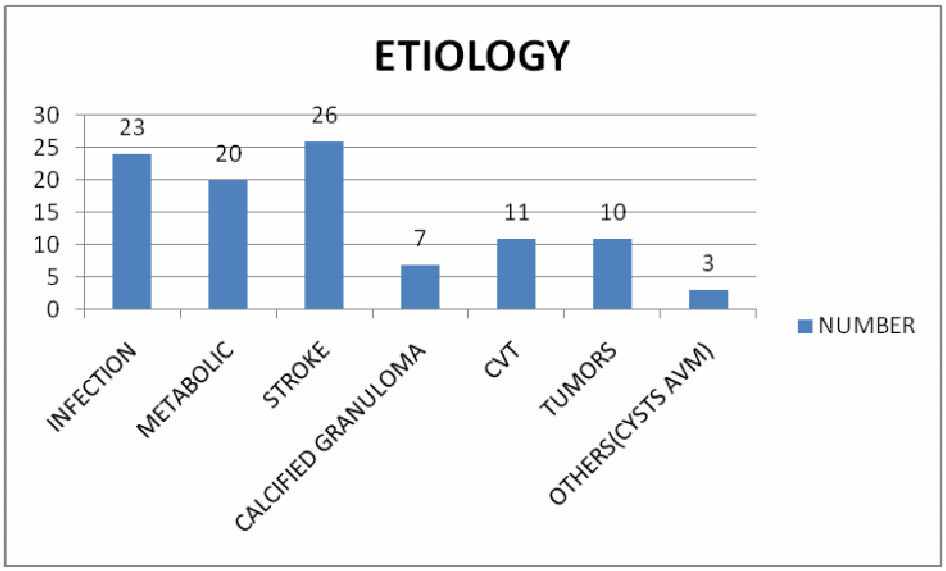


Out of 100 patients with acute symptomatic seizures 39 patients had focal neurological deficit. Hemiplegia was there for 29 patients. Monoplegia was there for 3 patients. Hemianopia and cranial nerve palsy was present for 6 patients. All these patients weakness persisted for more than 5 days of hospital stay. None of these patients had Todd's palsy.

All baseline investigations done for all patients.Out of 100 cases 8 patients had hyperglycemia and 7 had hypoglycemia. Both hypoglycemia and hyperglycemia resulted in seizures and controlled with correction of glycemic status . There was 10 patients with elevated urea and creatinine levels,of these 3 had urea >90mg/dl , 7 had <90 mg/dl, and creatinine >8mg /dl for 3 patients, <8mg /dl for 7 patients . All these patients had features of uremia and seizure controlled with AED followed by dialysis. Out of 100 cases 5 patients had hyponatremia with Na values <118 meq/l .Out of these 5 patients 3 had other associated metabolic abnormalities and 2 patients seizure was purely due to hyponatremia . LFT was abnormal in 3 patients. Chest X-ray was abnormal in 15 patients. Out of 15 patients 5 had mass lesion, 7 had tuberculosis and 3 had cardiomegaly. ECG was normal in 80 patients left ventricular hypertrophy was there for 13 patients ,features of coronary artery disease was there for 7 patients.

TABLE 9: Underlying Cause

UNDERLYING CAUSE	NUMBER	PERCENTAGE
INFECTION	23	23
METABOLIC	20	20
STROKE	26	26
CALCIFIED GRANULOMA	7	7
CVT	11	11
TUMORS	10	10
OTHERS(CYSTS AVM)	3	3

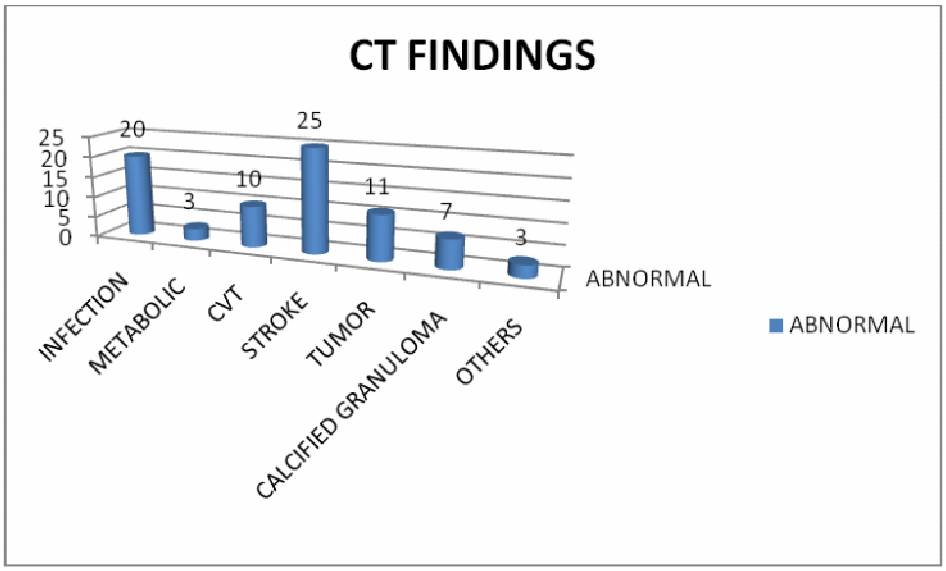


Out of 100 cases 26% of cases were due to Stroke, 20% metabolic causes, 23% infection,10% cases tumor ,11% cases were CVT ,7 % cases were single calcified

lesions .3% were classified as others 1 arachnoid cyst, 1 AVM, 1 meningomyelocele.

TABLE 10: Underlying Cause and CT Scan findings

CT SCAN FINDINGS	INFECTION	METABOLIC	CVT	STROKE	TUMOR	CALCIFIED GRANULOMA	OTHERS
NORMAL	3	17	1	-	-	-	-
ABNORMAL	20	3	10	26	10	7	3



CT brain was taken for all cases of acute symptomatic seizures. It was normal in 22% of cases. It was normal in 17 cases of metabolic causes of seizures and abnormal in 3 cases. Showed abnormal report in all cases of stroke, tumor and calcification.In patients With CNS infections 84% showed abnormal report. In

3% of cases of acute symptomatic seizures CT was normal and MRI was abnormal . In case of CVT 90% of times it was abnormal .Out of 100 patients 12 showed hemorrhage, 20 showed Infarct, 4 showed Tuberculoma, 3 Neurocysticercosis, 6 Tumour primary, 4 Secondary, 3 Abscess, of the 3 cases of abscesses 2 showed CSOM with CVT , 5 Ring enhancing lesions, 10 Calcification, 6 CVT, 1 CSOM ,arachnoid cyst 2 cases and diffuse cerebral oedema was present in 5 cases . MRI brain was taken for 35 patients. MRI was not taken for metabolic cases and for patients whose CT brain is diagnostic. All the 35 patients CT was inconclusive and the diagnosis could not be made .There was 4 cases with Tuberculoma , 3 cases of Neurocysticercosis , 1case of brain abscess , 7 cases of encephalitis and 5 case of meningitis . Patient with mass in the CT brain 2 showed primary brain tumor and 3 showed mutiple secondaries. Out of 35 cases 11cases of CVT was there.

CT brain was normal in 4 patients and MRI was abnormal in these 4 cases two had CVT , one had hemorrhage and another one had encephalitis.

Table 11: Underlying Cause and Age in percentage

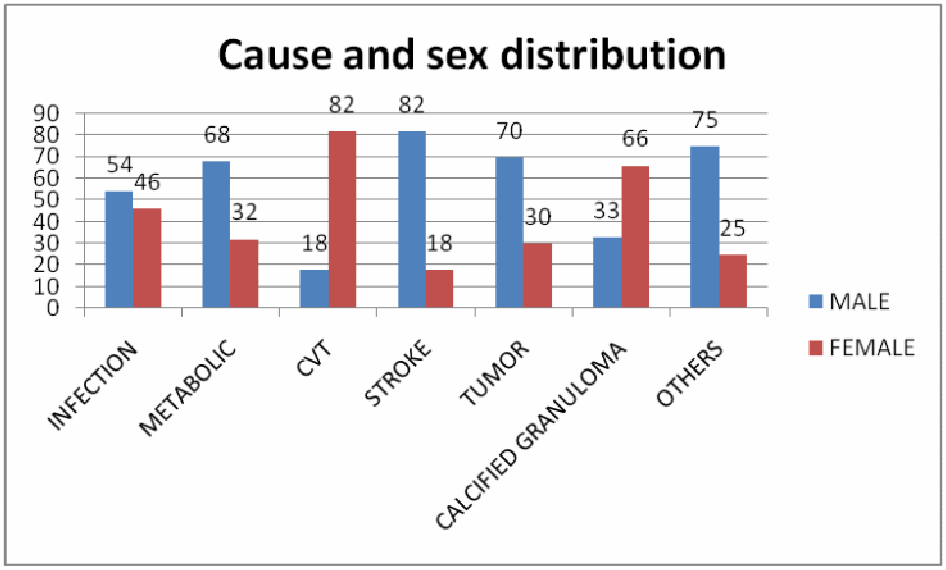
AGE GROUP	INFECTION	METABOLIC	CVT	STROKE	TUMOR	CALCIFIED GRANULOMA	OTHERS
UPTO20 YRS	50	10	-	-	-	30	10
21-30 YRS	36	4	28	-	12	16	4
31-40 YRS	40	20	5	30	5	-	-
41-50YRS	19	27	9	36	9	-	-
51-60YRS	-	18	-	64	18	-	-
>60 YRS	-	40.9	4.5	36	18.18	-	-

Patients up to 20 years of age most common cause was infections. Young adults from 20-30 years infection was most common followed by CVT and calcified granuloma. In patients with 30-40 age group most common cause was infection followed by stroke and metabolic. Patients aged 41-50 stroke was followed by metabolic causes. Patients aged 51-60 most common etiology was stroke and next common was tumor and metabolic .Patients >60 years metabolic causes were leading followed by stroke and tumors .Infections were common in younger patients,metabolic abnormalities were the common causes in older adults and

elderly . CVT and single calcified lesion was common in 20-30 years of age. Stroke and tumors common in patients >50 years of age.

TABLE12; CAUSE AND SEX DISTRIBUTION

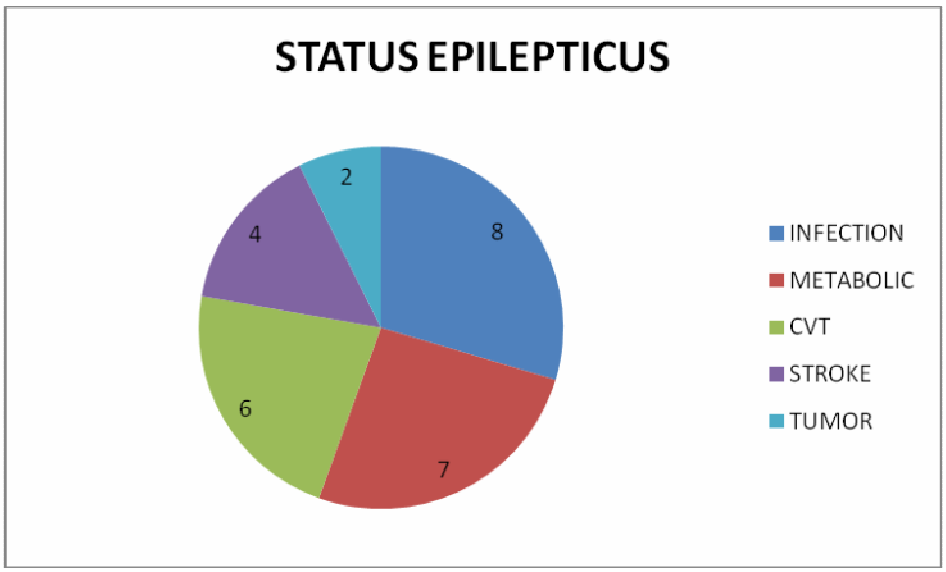
CAUSE	MALE (%)	FEMALE (%)
INFECTION	54	46
METABOLIC	68	32
CVT	18.1	81.9
STROKE	82	18
TUMOR	70	30
CALCIFIED GRANULOMA	34	66
OTHERS	75	25



Most of the causes were common in males than females except CVT. It was common in female. 81.9% of CVT occurred in females, In that postpartum CVT(55%) .

TABLE 13; CHARACTERISTICS OF THE 27 CASES OF STATUS EPILEPTICUS

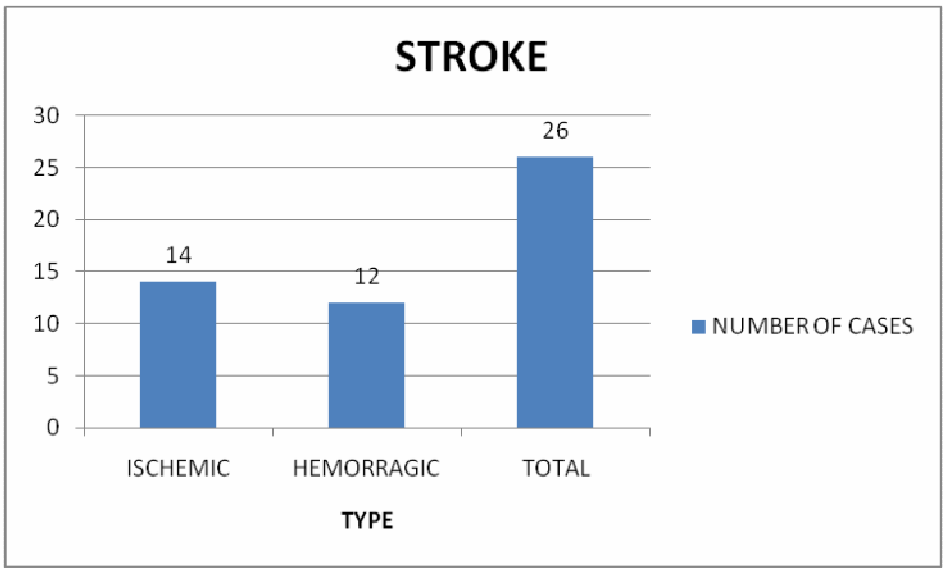
UNDERLYING CAUSE	FREQUENCY	PERCENTAGE
INFECTION	8	29.6%
METABOLIC	7	25.9%
CVT	6	22.2%
STROKE	4	14.8%
TUMOR	2	7.4%
TOTAL	27	100



Status epilepticus was the presentation in 27 cases .Most common cause of status epilepticus was CNS infections followed by Metabolic. 6 patients with status epilepticus there was CVT. Out of 27 cases there were 17 males and 10 females.

TABLE 14: ETIOLOGY OF ACUTE SYPTOMATIC SIEZURES STROKE-MOST COMMON CAUSE

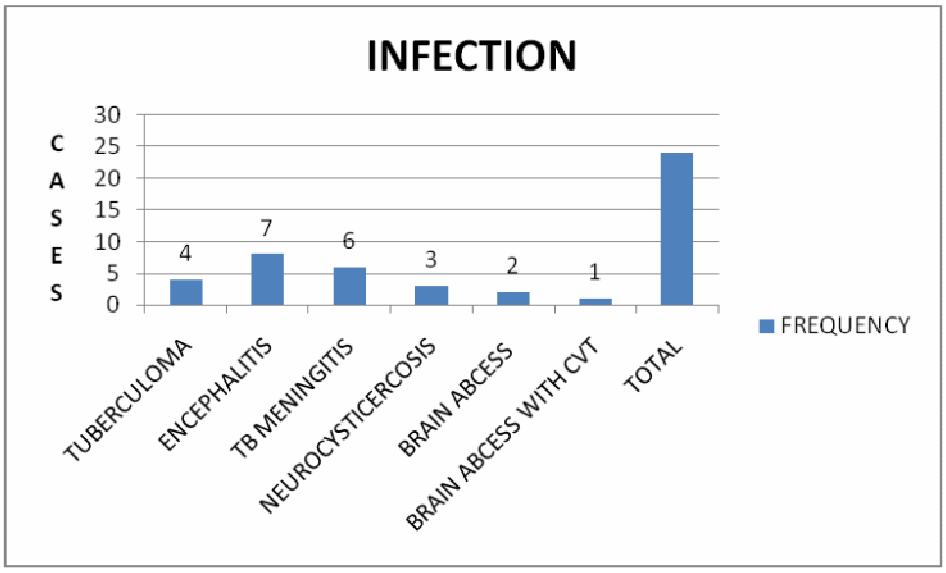
TYPE OF STROKE	NUMBER OF CASES	PERCENTAGE
ISCHEMIC	14	53.8%
HEMORRAGIC	12	46.15%
TOTAL	26	100%



Out of 26 cases of stroke 14 cases were due to ischemic stroke and 12 cases were due to hemorrhagic stroke.

TABLE 15: ETIOLOGY OF ACUTE SYPTOMATIC SIEZURES INFECTION -SECOND MOST COMMON CAUSE

DIAGNOSIS	FREQUENCY	PERCENTAGE
TUBERCULOMA	4	17.3%
ENCEPHALITIS	7	30.4%
TB MENINGITIS	6	26%
NEUROCYSTICERCOSIS	3	13%
BRAIN ABCESS	2	8.6%
BRAIN ABCESS WITH CVT	1	4.3%
TOTAL	23	100%



Out of 23 cases of infection 4 cases (17.3%) were due to Tuberculoma, 7(30.4%) cases were due to encephalitis, 6(26%) cases were due to TB meningitis 3(13%) cases were due to neurocysticercosis,2 cases of brain abcess, 1 case with brain abcess with CVT. Out of 20 cases of metabolic cause of seizures 5 were due to hypoglycemia,6 were due to hyperglycemia,5 cases were due to hyponatremia,2 cases were due to hypocalcemia, 2 cases with uremia. Out of 100 cases there were 11 cases of CVT and 1 cases of CVT with brain abscess secondary to CSOM. Of these 2 were males (18%) and 9 were females (82%). Out of 9 females 5 were postpartum CVT (55%).

There was 7 cases of single calcified granuloma out of 100 patients of acute symptomatic seizures. It was most common in patients from 21 to 30 years of age.

There were 10 cases of tumors as a cause of acute symptomatic seizures. Of these 6 were primary brain tumors and 4 were secondary tumors. Of these secondary tumors,all 4 had carcinoma lung . Out of 100 cases, 3 cases classified as others,out of that 1 had arachnoid cyst ,1 had meningomyelocele with ventricular dilatation and 1 had AV malformation.

DISCUSSION

Annal Gandhi Memorial Government Hospital, Trichy is the tertiary referral care hospital located in Trichy. Various cases have been referred from Government sector hospitals like PHCs, Taluk hospitals, District head quarters hospitals, ESI hospital and many private hospital of not only from Trichy but also from near by districts. Study of acute symptomatic seizure patients from June 2010 to Oct 2011 included 100 patients.

DEMOGRAPHY

Acute symptomatic seizure is an important cause of morbidity in our part of the country. It is very important to find out the underlying cause and its treatment for prevention of recurrent seizure. Acute symptomatic seizure is different from epileptic syndromes as it is curable by treating the underlying cause . Even if AEDs are used to suppress recurrence of seizures, they generally do not need to be continued after the patient has recovered from the primary illness. These concepts are based on the basic assumption that acute symptomatic seizures presumably cease with the resolution of the precipitating cause or illness. But seizure can recur in conditions like tumor ,stroke and head injury .

There are 63 cases of males and 37 cases of females in our study. The mean age is 40 and there is patients from 13 to 91 years of age.

Study by Sander et al[26] the proportions of males and females were similar.

Usha Kant Misra et al's study the median age of the patients was 37 years from 16-78 years.

According to Jaishree T Narayanan et al the mean age of patients with acute symptomatic seizures was 49.07+20.29 years (six months to 80 years) as they had included pediatric patients in their study .In our study nearly half of patients are in the age group of 21 to 40 years ,11% of patients are less than 20 years and 22% are more than 60 years .Study by Sander et al 25% (21-28%) were younger than 15 years and 24% (21-28%) were 60 years or older. Twenty-four (36%) were aged 60 years and above in Jaishree T Narayanan et al study.

MODE OF PRESENTATION

In our study 54% of patients the type of seizure is GTCS, 1/3 presented with focal seizure,11% focal with secondary generalisation and and 2% with epilepsia partialis continua . Our study correlates with previous studies. Study by Usha Kant Mishra et al generalized tonic-clonic seizure was the seizure type in 36 (55%) patients and in the remaining 30 (45%) patients, the seizure type was partial with or without secondary generalization. Of the 30 patients with partial seizures, 28(93%) had complex partial seizure and two (7%) had epilepsia partialis continua in their study. Study by Clifford Schold a total of 56% of the patients had focal motor seizures, and in 44%, the seizures were generalized. According to Sridharan et al[3] in the new cases of epilepsy 50% have seizures of partial origin and 50% of generalized origin before the age of 40 years. After 40 years, the proportion of partial epilepsy rises to 75% by the age of 75.

Study by J. M. K. Murthy & Ravi Yangala[5] type of seizure was simple partial or complex partial with or without secondary generalization in 412 (78%)patients and either unlocalized or generalized in 114 (22%) patients.

Status epilepticus is present in 27% our patients. Most common cause for status epilepticus is infection(29.6%) followed by Metabolic disturbances(25.9%) and CVT (22.2%). According to Jaishree T Narayanan et al 10 (15%) patients had seizure clusters and four (6%) patients presented with SE. Usha Kant Misra et al 's study 35 patients had convulsive status epilepticus, and 2 patients had nonconvulsive status epilepticus .

ETIOLOGY

Most common cause of siezure in our study is Stroke followed by infection followed by metabolic causes. Infection is the cause of siezure in nearly ¼ of the patients .Our study correlate with other studies from south india 3,[4,5,12]Out of 100 cases 26% of cases are due to stroke , 23% due to infection causes, 20% due to metabolic , 10% due to tumor , 11% due to CVT and 7% are due to single calcified lesions . 3% are classified as others include 1arachnoid cyst,1AVM, 1 meningomyelocele with hydrocephalus .

Another study from south India by Jaishree T Narayanan[12] showed central nervous system (CNS) infections in nearly 1/3 of patients. Study by J. M. K. Murthy et al seizure occurred in close temporal association with an acute central nervous system (CNS) insult in 53% of patients. Infections of CNS including single CT enhancing lesion accounted for 77% of patients with acute

symptomatic epilepsy. Study by Ravindra Kumar Garg et al[23] infective pathologies were the most common etiology.

In our study out of 23 cases of infections 6 cases are due to TB meningitis,4 cases are due to Tuberculoma , 3 cases of neurocysticercosis, 8 cases of encephalitis, 2 cases of brain abscess and 1 cases of brain abscess with CVT secondary to CSOM are there. The distribution of the pathology according to Jaishree T Narayanan et al[12] in patients with CNS infections was meningoencephalitis in 43% and parenchymal granuloma in 57% of patients out of that 75% due to degenerative phase solitary cystic granuloma and 25% due to tuberculoma . Study by J. M. K. Murthy et al Neurocysticercosis, SCTEL and small single cerebral calcific CT lesion (SSCCCTL) together accounted for 40% of etiological factors and Neurotuberculosis for 10%. In our study Neurotuberculosis accounts for 41.6% of infections and according to Jaishree T Narayanan et al it was 25% and J. M. K. Murthy et al 10%. Study by Ravindra Kumar Garg et al[23] tuberculosis was the commonest infective pathology. Usha Kant et al 's study there was 5 cases of Tuberculous meningitis and 5 cases of Tuberculoma brain was there. Brain tuberculomas make up 5 to 8 per cent of intracranial masses in person in developing countries[18]Before effective chemotherapy was available for tuberculosis, tuberculoma made up 20 per cent of intracranial lesions in one large series.[19]In our study 3 cases are due to Neurocysticercosis and 7 cases due to small single cerebral calcific CT lesion . According to Jaishree T Narayanan et al parenchymal granuloma in 57% patients

with CNS infections ,out of that 75% due to degenerative phase solitary cystic granuloma. Study by J. M. K. Murthy et al neurocysticercosis, SCTEL and small single cerebral calcific CT lesion (SSCCCTL) together accounted for 40% of etiological factors. Study by Ravindra Kumar Garg et al[23] second most common infection was neurocysticercosis following Tuberculosis. According to Thussu et al [45] solitary cysticercus granuloma, a benign form of parenchymal neurocysticercosis, is considered to be the most common aetiology for SSECTL . According to Macro et al[44] neurocysticercosis is the most common parasitic disease of the CNS in developing countries. According to Montano et al an important proportion of seizure cases are associated with Neurocysticercosis in endemic areas.

In our study 1/5 of the cases are due to metabolic causes. Jaishree T Narayanan et al [12]study showed metabolic disorders in nearly 1/3 of the cases of seizures. In our study there are 9 cases of uremia , 8 cases of hyperglycemic hyperosmolar state, 5 cases of hypontremia and 7 cases of hypoglycemia,2 cases of hypocalcemia . Study by Jaishree T Narayanan[12]out of 21 cases 15 were due to hyponatremia, 4 were due to hyperglycemia, 2 were due to hypoglycemia. Study by Jaishree T Narayanan[12] there was 6 cases of alcohol related seizures and 2 cases of hypoxic encephalopathy

In our study 26% of cases the cause of seizure is stroke and 11% due to CVT, Out of these 14 cases(53.8%) are due to ischemic stroke , 12 cases are (46.15%) related. to Hemorragic Stroke. By Jaishree T Narayanan[12]

cerebrovascular diseases (ischemic, venous and hemorrhagic) in 21% cases and by JMK Murthy vascular in 14% (Ischemic 6%, Haemorrhagic 5% , CVT 3%) of cases .Mean age of patient presenting with seizure due to stroke was 48+/-18 years . Studies from developed countries acute symptomatic seizure due vascular causes were common. Study by Sander et al[26] vascular disease in 15% (12-18%), among older subjects 49% (41-58%) were due to vascular disease. Study by J. M. K. Murthy et al cerebrovascular diseases were the risk factors in 48% of patients with remote symptomatic epilepsy and cerebrovascular diseases were the etiological factors in 64% of patients aged >40 years . By Ettinger AB et al the most common single cause of seizures was infarction or hemorrhage (54%). According to Sridharan et al cerebrovascular disease is the most commonly identified cause among adults, 37% of symptomatic seizures. There are 11 cases of CVT and 1 case of CVT with brain abscess secondary to CSOM in our study. Out of these 9 patients are females (81.8%) and 2 patients are males (18.1%) . Out of 9 females 5 are postpartum CVT (55.5%). CVT is a common cause of seizure in the postpartum period in our hospital. Jaishree T Narayanan et al study 3% of cases were due to CVT. Study by Dr J. M. Murthy, Department of Neurology, Nizam's Institute of Medical Sciences, Hyderabad 3% of cases were due to CVT. Cortical sinovenous thrombosis is an important cause of acute symptomatic seizures among young patients with cerebrovascular diseases . A number of conditions have been etiologically linked to cortical sino

venous thrombosis. But in India the majority of cases are related to pregnancy and purperium.

Seizure associated with fever headache or vomiting should be investigated to find out the underlying cause . In our study patients with infections and CVT had headache,vomiting and fever. Only 22 patients had fever at the time of seizures. Patients with meningitis, brain abscess, encephalitis were presented with fever . Headache and vomiting was present in around 50 patients. Most of these patients had mass lesions like tumor neurocysticercosis,tuberculoma abscess or CVT. In our study 39 patients had neurological deficit, most of the cases due to CVT , tumor or stroke .Cerebral AVM and arachnoid cyst are other rare causes for seizure. For the diagnosis of AVM , MRI brain is the investigation of choice . Immediate non-contrast CT is useful for emergency patients presenting with seizure to guide appropriate acute management, especially where there is an abnormal neurologic examination, predisposing history, or focal seizure onset. MRI has been shown to be highly sensitive and specific in identifying the underlying pathology in partial epilepsy. MRI may determine patient selection for surgery and directly affects the presurgical evaluation and operative strategy. Therefore MRI should be performed early to avoid unnecessary medication in patients with resectable intracranial mass lesions.

Seizures may herald or complicate acute neurological and medical disorders.The etiological spectrum in the present study was distinctly different when compared to the data from developed countries and it well correlate with

other studies from developing countries and other studies from south India. New-onset acute symptomatic seizures are different from unprovoked seizures in that they generally do not recur and usually do not need long-term AED therapy. However, this study suggests that the risk of seizure recurrence or SE after the first seizure is likely to be high in patients with acute focal cerebral lesions and diffuse CNS infections like meningoencephalitis and encephalitis. Of the patients who had seizure recurrence or developed SE, in 38% the pathology was infection-related and the other commonality was cortical gray matter involvement. Probably this group of patients, particularly patients with CNS infections, with high risk of seizure recurrence may need AED prophylaxis, at least for the period of resolution or stabilization of acute CNS insult.

When considering the results of this study the limitations of the study must be recognized. This is a highly selected population and the findings may not be generalizable. In developing countries CNS infections like Japanese encephalitis, Tuberculous meningitis, bacterial meningitis and NCC are endemic and are frequent risk factors for new-onset acute symptomatic seizures. There is a need to study a large population of patients with these pathologies for the risk of recurrence of seizures as it may have therapeutic implications, possible AED prophylaxis.

SUMMARY

The study "A STUDY OF PATIENTS PROFILE WITH ACUTE SYMPTOMATIC SEIZURES IN ANNAL GANDHI MEMORIAL GOVERNMENT HOSPITAL, TRICHY " was a cross sectional study of 100 patients admitted with first episode of seizures in Annal Gandhi Memorial Government Hospital Trichy . Patients who satisfied the inclusion criteria were included in the study and a detailed history was taken from the relatives about the type of seizure and co morbid conditions. Examination of CNS was done to find out any under lying neurological deficit including fundus examination to look for papilledema.Investigations done in all patients were CBC, blood sugar , urea , creatinine ,serum electrolytes ,liver function test ,ECG , chest x-ray and CT brain was done for all patients. MRI brain was done for some indicated cases. Analysis of the type of seizure, etiology of seizure and specific pathology was done. This study shows that CNS infections like Tuberculoma , Neurocysticercosis , encephalitis and brain abscess are treatable causes of seizures in our hospital . So identification of the underlying pathology by various investigations and neuroimaging modalities are very important in the management of these patients. Patients with metabolic abnormalities, on correction of the underlying condition seizure will be controlled and diagnosis is important to avoid long term AED treatment.

CONCLUSION

- Acute symptomatic seizure can develop at any age and it is most common in patients 20-40 years of age followed by >60 years.
- Most common cause of acute symptomatic seizure is Stroke in our patients. Second most common cause is CNS Infections and following that Metabolic ,CVT , and tumors of brain both primary and secondary and calcified granuloma .
- Infections and single calcified lesions are common in young patients and metabolic conditions are common in older patients.
- CVT is an important cause of seizure in females especially during peripartum period. In males CVT is secondary to infection.
- In stroke ischemic stroke is most common cause of seizures followed by hemorrhagic stroke.
- Meningoencephalitis and Tubercular meningitis are the most common CNS infections causing seizures followed by Tuberculoma brain and neurocysticercosis.
- Metabolic causes of seizures are most common with uremia followed by hyperglycemia and hypoglycemia and other cases are due to hyponatremia, hypocalcemia.
- Immediate non-contrast CT is useful for emergency patients presenting with seizure to guide appropriate acute management, especially where there

is an abnormal neurologic examination, predisposing history, or focal seizure onset.

- MRI has been shown to be highly sensitive and specific in identifying the underlying pathology in partial epilepsy.

BIBLIOGRAPHY

1. Harrison's Principles of Internal Medicine, 18th edition
2. Adams and Victor's principles of neurology,9th edition
3. R.Sridharan; CURRENT 664 SCIENCE, VOL. 82, NO. 6, 25 MARCH 2002; Epidemiology of epileps. Department of Neurology, Apollo Hospitals
4. J. M. K. Murthy and Y. V. S. Reddy ; Journal of Neurological Sciences 159 (1998), pp. 151–155; Prognosis of epilepsy associated with single CT enhancing lesion, a long term follow-up study.
5. Murthy J M K; Seizure. 1999 May;8 (3):162-5;.Acute symptomatic seizures - incidence and etiological spectrum: a hospital-based study from South India. Department of Neurology, Nizam's Institute of Medical Sciences
6. V Arora, IBS Nijjar, DS Mahajan, PS Sandhu, JP Singh, R Chopra From Nijjar Scan & Diagnostic Centre, Amritsar, India; NEURORADIOLOGY Year : 2005; Volume : 15 ;Issue : 3;| MRI in seizure disorder - a pictorial assay
7. API text book of medicine, 9th edition.
8. Claassen J, Mayer SA, Kowalski RG, Emerson RG, Hirsch LJ;Detection of electrographic seizures with continuous EEG monitoring in critically ill patients.Division of Critical Care Neurology, Department of Neurology, College of Physicians and Surgeons, Columbia University.

9. J MK Murthy, T Jayashree Naryanan;Continuous EEG monitoring in the evaluation of non-convulsive seizures and status epilepticus.

10. Byron Bernal MD and Nolan R. Altman MD; Neuroimaging Clinics of North America Volume 13, Issue 2, May 2003 Evidence-based medicine: neuroimaging of seizures Department of Radiology, Miami Children's Hospital.

11. Parmar H, Lim SH, Tan NC, Lim CC. +Neurology 2006; 66:1732–1735]; Acute Symptomatic Seizures and Hippocampus Damage: DWI and MRS Findings.

12. Jaishree T Narayanan, JMK Murthy ; NEUROLOGY INDIA Year : 2007/Volume : 55Issue : 2| Page : 136-140;New-onset acute symptomatic seizure in a neurological intensive care Unit, Department of Neurology,The Institute of Neurological Sciences, CARE Hospital

13. A NORIEGA-SANCHEZ; NEUROLOGY 1978;28:667 Clinical and electroencephalographic Changes in progressive uremic encephalopathy

14. Sunil PRADHAN, Ramakant YADAV ;Neurology Asia 2004; 9 Seizures and epilepsy in central nervous system infections Department of Neurology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, India.

15. H. Del Brutto; Acta Tropica Volume 87, Issue 1, June 2003, Imaging findings in Neurocysticercosis ; Department of Transmissible Diseases, Instituto Nacional de Ciencias Neurologicas.

16. J. M. K. Murthy and Y. V. Subba Reddy; Prognosis of epilepsy associated with single CT enhancing lesion: A long term follow up study Department of Neurology, Nizam's Institute of Medical Sciences.

17. Florian Mormann et al;Review Articles Seizure prediction: the long and winding road .Department of Epileptology, University of Bonn Bonn.

18. Bouchama A, Al-Kawi MZ, Kannan I: Brain biopsy in Tuberculoma: The risks and benefits. Neurosurgery 1991, 28:405.

19. Rammamurthi B, Varadarajan M: Diagnosis of Tuberculoma of the brain. J Neurosurg 1961, 18:1.

20. Sheller J R, des Prez R M: CNS tuberculosis. Neurol clin 1986,4:143.

21. M. N. A. Idris et al; INT J TUBERC LUNG DIS 11(1):91–95 2007 The Union. Tuberculoma of the brain: a series of 16 cases treated with antituberculosis drugs.

22. Poonnoose S I, Rajshekhar V. Rate of resolution of histological verified intracranial tuberculoma. Neurosurgery 2003; 53: 873–89.

23. Ravindra Kumar Garg , Parthiv Desai, Minakshi Kar and Alok Mohan Kar Department of Neurology, King George's Medical University Multiple ring enhancing brain lesions on computed tomography: An Indian perspective.

24. Shobha N ; Neurology India, Vol. 54, No. 4, October-December, 2006,pp. 440-442 ; Brief Report Diabetic nonketotic hyperosmolar state: Interesting imaging observations in 2 patients with involuntary movements and seizures.

25. Wasterlain CG.Epilepsia partialis continua.In:Seizures andEpilepsy.e.Medicine.com Inc: May 4, 2006.

26. Sander JW, Hart YM, Johnson AL, Shorvon SD; Lancet. 1990 Nov 24; 336(8726):1267-71. National General Practice Study of Epilepsy: newly diagnosed epileptic seizures in a general population

27. Ravindra Kumar Garg, Manish Kumar Singh and Surendra Misra; Epilepsy ResearchVolume 38, Issues 2-3, February 2000, Pages 91-104; Single-enhancing CT lesions in Indian patients with seizures: a review. Department of Neurology,Institute of Medical Sciences, Banaras Hindu University.

28. Commission on Epidemiology and Prognosis,International League Against Epilepsy. Epilepsia 1993;34:592-6.

29. Bleck TP, Smith MC, Pierre-Louis SJ, Jares JJ, Murray J;Crit Care Med. 1993 Jan;21(1):Neurologic complications of critical medical

illnesses.Department of Neurology, University of Virginia School of Medicine.

30. Varelas PN, Mirski MA; J Neurosurg Anesthesiol. 2001 Apr; 13(2); Seizures in the adult intensive care unit. Neurosciences Critical Care Unit, The Johns Hopkins Hospital, Baltimore, Maryland 21287, USA.

31. Timmons S, Sweeney B, Hyland M;Ir Med J. 2002 Feb;95(2):47-9; New onset seizures in the elderly: aetiology and prognosis.

32. Wijdicks EF, Sharbrough FW; Neurology. 1993 May;43(5):1042-4;New-onset seizures in critically ill patients.Department of Neurology, Mayo Clinic, Rochester.

33. Nagaraja D, Taly AB, Puerperal venous sinus thrombosis in India –In: Sinha K.K. ed. Progress in clinical neurosciences, Ranchi, NSIpublication 1989; 5: 165-177

.34. Seizures in patients with supratentorial oligodendroglial tumours Acta Neurochirurgica Volume 135, Numbers 1-2 / March,1995.

35. Epilepsia Volume 44 Issue 9, Pages 1223 – 1232; Published Online: 12 Aug 2003 Brain Tumor and Seizures.

36. Authors: John Tattersall;Seizure activity post organophosphate exposure Journal: Frontiers in Bioscience

.37. Chopra JS, Banerje AK. Primary intracranial sino venous occlusions in

youth and pregnancy. In: Vinken PJ , Bruyn GW, Klawans HL, Toole JK, eds. Vascular disease part II. Amsterdam: Elsevier science group 1989: pp 425 – 452.

38. Isensee CH, Raul J, Thron A. Magnetic resonance imaging of thrombosed dural Sinuses. Stroke 1994; 25(1): 29.

39. Stam J: Thrombosis of the cerebral veins and sinuses. N Engl J Med 2005; 352:1791–1798.

40. Singhvi JP, Sawhney IM, Lal V, Pathak A, Prabhakar S;Profile of intractable epilepsy in a tertiary referral center. Department of Neurology,Postgraduate Institute of Medical Education and Research, Chandigarh.

41. Carpio A, Hauser WA; Curr Neurol Neurosci Rep. 2009 Jul;9(4):319-26; Epilepsy in the developing world..Research Institute, School of Medicine, University of Cuenca, Ciudadela Universitaria, Cuenca, Ecuador.

42. Dale C. Hesdorffer and Marco D'Amelio GH Sergievsky; Epilepsia Volume 46 Issue s11, Pages 43 - 45 ;Mortality in the First 30 Days Following Incident Acute Symptomatic Seizures Center and Department of Epidemiology, Mailman School of Public Health, Columbia University.

43. Pradeep P. Nair, Jayantee Kalita and Usha K. Misra; European Journal of Radiology Role of cranial imaging in epileptic status Department of

Neurology, Sanjay Gandhi Post Graduate Institute of MedicalSciences, Lucknow .

44. MARCO T. MEDINA, M.D., AND CHRISTOPHER M. DEGIORGIO, M.D; Neurosurg Focus 12 (6): Introduction, 2002 . Introduction to neurocysticercosis: a worldwide epidemic.Department of Neurology, University of California at Los Angeles, Sylmar, California; Department of Neurology, National Autonomous University of Honduras.

45. A Thussu, A Chattopadhyay, I M S Sawhney, N Khandelwal ; Albendazole therapy for single small enhancing CT lesions (SSECTL) in the brain in epilepsy;Journal of Neurology, Neurosurgery, and Psychiatry 2008; 79:272-275.

46. Mitchell WG;Epilepsia. 1996;37 Suppl 1:S74-80. Status epilepticus and acute repetitive seizures in children, adolescents, and young adults: etiology, outcome, and treatment..Department of Neurology and Pediatrics, University of Southern California School of Medicine, Los Angeles, USA.

47. Bleck TP, Smith MC, Pierre-Louis SJ, Jares JJ, Murray J, Hansen CA; Crit Care Med. 1993 Jan;21(1):Neurologic complications of critical medical illnesses. Department of Neurology,University of Virginia School of Medicine.

48. Brains diseases of nervous system; 11th edition .

49. Ettinger AB, Shinnar S; Neurology 1993, Mar;Neurology 1994 Jan;44(1): New-onset seizures in an elderly hospitalized population.

50. Ravindra Kumar Garg ;Epilepsy ResearchVolume 38, Issues 2-3, February 2000, Pages 91-104;Single-enhancing CT lesions in Indian patientswith seizures: a review , Department of Neurology, Institute of Medical Sciences, Banaras Hindu University.

51 V. Rajashekar; Acta Neurolgica Scandinavica 84 (1991), pp. 465–470; Etiology and management of single small CT lesion in patients with seizures. Understanding controversy.

52. P. R. M. Bitterncourt, B. Admolekum and N. et al; Baruch Epilepsia 37 (1996), pp. 1128–1137;, Epilepsy in the tropics II: clinical presentations, pathophysiology, immunologic diagnosis; economics and therapy.

53. J. F. Annegers, W. A. Hauser, J. R. J. Lee and W. Rocca Epilepsia 36 (1995), pp. 327–333;, Incidence of acute symptomatic seizures in Rochester, Minnesota, 1935–1984.

54. Salahudeen MM, Inbasekaran V, Kumar NA, Rajan DK, Sivakozhundu MK Neurology India Year: 2001 Otogenic intracranical suppuration at a rare site .

55. K. Haddadian et al;.The Internet Journal of Infectious Diseases. 2005 Volume 4: Multiple Brain Tuberculomas And Role Of Open Brain

Biopsy: A Case Report And Review.

56. J.M.K. Murthy and V. Sreenivas Reddy; SeizureVolume 7, Issue 2, April 1998, Clinical characteristics, seizure spread patterns and prognosis of seizures associated with a single small cerebral calcific CT Department of Neurology, Nizams Institute of Medical Sciences

57. Nicoletti Aet al; Epilepsia. 2009 Jun26. Natural history and mortalityof chronic epilepsy in an untreated population of rural Bolivia: A follow-up after 10 years.Department of Neurosciences, University of Catania.

58. Dale C. Hesdorffer ; Epilepsia Volume 50 Issue 5, Pages 1102 - 1108 26 Jan 2009. Is a first acute symptomatic seizure epilepsy? Mortality and risk for recurrent seizure .Department of Epidemiology.

59. European Journal of Emergency Medicine: February 2008 - Volume 15 Seizures and hyponatremia after excessive intake of diet coke.

60. Koppel BS et al; Curr Treat Options Neurol. 2009 Jul;11(4):231-41 ; Treatment of acute and remote symptomatic seizures.

61. J. F. Annegers, W. A. Hauser, J. R. J. Lee and W. Rocca, Incidence of acute symptomatic seizures in Rochester, Minnesota, 1935–1984. Epilepsia 36 (1995), pp. 327–333.

62. Kim, M. A, Park, K. M, Kim, S. E ; European Journal of Neurology,Volume 15,Number 1,January 2008;Acute symptomatic seizures

in CNS infection .

63. Usha Kant Misra MD., DM, Jayantee Kalita MD,DM and Pradeep P. Nair MD The American Journal of MedicineVolume 121, Issue 7, July 2008 Status Epilepticus in Central Nervous System Infections: An Experience From a Developing Country .Department of Neurology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, India.

64. Varelas, Panayiotis N. MD, PhD; The Neurologist: May 2006 - Volume 12 – Issue 3 - pp 127-139; Management of Seizures in the Critically Ill.

65. D Goel, KK Bansal, A Singhal, R Srivastav; JAPI • VOL. 56 • DECEMBER 2008; Two-tier System of Epilepsy Evaluation: A Useful Method for Developing Countries.

66. Leppik IE: Contemporary Diagnosis and Management of the Patient with Epilepsy, Fifth Edition. Newtown, Pa: Handbooks in Health Care; 2000.

67. Liporace JD, Sperling MR: Simple autonomic seizures. In: Engel J Jr, Pedley TA, eds. Epilepsy: A Comprehensive Textbook. Philadelphia, Pa: Lippincott-Raven; 1997: 549-555.

PROFORMA

Name: Income

Age: occupation

Sex: education:

Residence: socioeconomic status

COMPLAINTS:

Type of seizure:

Status epilepticus::

H/O vomiting:

H/O loss of consciousness:

H/O fever

H/O headache

H/O weakness:

H/O joint pain/ rash

H/O diarrhea

H/O trauma

H/O CSOM

Post partum state

H/O exanthematous illness

PAST ILLNESS

Diabetes

Hypertension

Meningitis

Tuberculosis

Malignancy/ Rx for malignancy

Alcohol use

Family H/O seizure

H/O HIV infection

H/o CKD

PERSONAL HISTORY

Pork eating

Contact with PT

Level of sanitation

H/O abortions

Tobacco use

EXAMINATION

Anemia

Cyanosis

Lympadenopathy

CNS:

HF:

Cranial nerves:

Fundus:

Focal deficit:

Other systems:

PR:

BP:

INVESTIGATIONS:

Hb TC DC ESR

Sugar urea creatinine

Na K Cl HCO3 Ca

LFT Bilirubin total direct indirect

AST ALT ALP

ECG

Carotid Doppler CSF:

CT/MRI

DIAGNOSIS:

MASTER CHART

SLN	A/S	SZR	FVR	HED	VMT	ALT	FND	PST	TRM	CSM	PI	PE H	BP	FUN	SUG	UR	Cr	SE	LFT	CXR	ECG	CT	MR	ETI
1	48/M	3	1	2	1	2	1	3	1	1	2	3	1	1	3	1	1	1	1	1	2	1	1	2
2	70/F	3	1	1	1	2	1	2	1	1	2	0	2	1	3	1	1	1	1	4	2	1	1	2
3	40/F	1	2	2	2	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	1	7	1	1
4	27/F	3	1	2	1	1	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	14	1	4
5	65/F	5	1	1	1	3	1	2	1	1	2	1	4	1	3	1	1	1	1	1	2	1	1	2
6	13/F	1	1	1	1	1	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	17	1	7
7	80/F	1	1	1	1	1	3	2	1	1	1	1	4	1	1	1	1	1	1	1	1	3	1	3
8	16/M	3	1	1	1	1	1	3	1	1	1	0	1	1	1	1	1	1	1	1	1	14	1	4
9	22/F	5	2	2	2	3	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	17	12	1
10	29/M	3	2	2	2	3	1	3	1	1	1	0	1	1	1	1	1	1	1	1	1	1	12	1
11	60/M	1	1	2	2	1	3	3	1	1	3	0	4	1	1	1	1	1	1	1	1	2	1	3
12	62/M	3	1	1	1	2	1	3	1	1	1	2	2	1	1	1	1	2	1	1	1	1	1	2
13	67/M	3	1	1	1	1	3	3	1	1	1	0	1	1	1	1	1	1	1	1	2	3	1	3
14	18/M	3	1	1	1	2	1	3	1	1	2	0	1	1	3	1	1	1	1	1	1	1	1	2
15	56/M	1	1	2	2	3	3	3	1	1	2	0	4	1	2	1	1	1	1	1	3	2	1	3
16	60/M	1	1	1	1	1	1	3	1	1	10	1	2	1	1	1	1	1	1	4	2	3	1	3
17	34/M	3	1	2	2	2	1	3	1	1	1	2	1	1	1	1	1	2	1	1	1	1	1	2
18	35/M	3	1	2	1	2	3	3	1	1	2	2	1	1	2	1	1	1	1	1	1	2	1	3
19	40/M	5	2	2	2	2	1	3	1	1	4	1	2	1	1	1	1	1	1	3	1	2	12	1
20	80/M	2	1	1	1	1	1	3	1	1	3	6	1	1	2	1	1	1	1	2	1	6	5	6
21	65/M	1	1	1	1	1	3	3	1	1	1	1	1	1	1	1	1	1	1	1	1	3	1	3
22	28/M	1	1	1	1	1	1	3	1	1	1	0	1	1	1	1	1	1	1	1	1	14	1	4
23	58/M	3	1	1	1	1	3	3	1	1	2	1	4	1	2	1	1	1	1	1	1	3	1	3
24	38/M	5	1	2	2	3	3	3	1	1	1	2	4	1	1	1	1	1	1	1	1	2	1	3
25	14/M	1	1	2	2	1	1	3	1	1	1	4	1	3	1	1	1	1	1	3	1	4	2	1

SLN	A/S	SZR	FVR	HED	VMT	ALT	FND	PST	TRM	CSM	PI	PEH	BP	FUN	SUG	UR	Cr	SE	LFT	CXR	ECG	CT	MR	ETI
26	13/M	1	1	1	1	1	1	3	1	1	1	0	1	1	1	1	1	1	1	1	1	17	1	7
27	30/M	5	2	2	2	2	3	3	1	1	1	0	1	1	1	1	1	1	1	1	1	14	8	1
28	22/M	5	1	2	1	2	1	3	1	1	1	2	3	1	1	1	1	1	1	1	1	8	6	5
29	65/M	1	1	1	1	3	3	3	1	1	1	1	1	3	1	1	1	1	1	2	1	6	1	6
30	45/M	5	1	1	1	3	1	3	1	1	1	0	1	1	1	1	1	1	1	1	1	13	1	6
31	39/F	1	1	2	2	1	3	2	1	1	1	1	1	1	1	1	1	1	1	1	1	13	4	6
32	21/M	1	1	2	1	1	1	3	1	1	1	3	1	1	1	1	1	1	1	3	1	4	2	1
33	48/M	2	1	1	2	3	3	3	1	1	3	0	4	1	1	1	1	1	1	1	2	2	1	3
34	75/M	3	1	1	1	1	3	3	1	1	10	0	1	1	1	1	1	1	1	1	1	3	1	3
35	22/M	5	2	2	2	3	1	3	1	1	1	O	2	1	1	1	1	1	1	1	1	3	8	1
36	43/M	3	1	2	1	1	2	3	1	1	1	2	4	1	1	1	1	1	1	1	1	3	1	3
37	45/M	1	1	1	1	1	2	3	1	1	1	0	4	1	1	1	1	1	1	1	3	3	1	3
38	35/M	2	1	2	2	2	1	3	2	1	1	2	1	1	1	1	1	1	1	1	1	2	1	3
39	60/M	1	1	1	2	2	3	3	1	1	3	2	4	1	1	1	1	1	1	1	2	2	1	3
40	22/M	1	1	1	1	1	1	3	1	1	1	0	1	1	1	1	1	1	1	1	1	13	1	6
41	38/M	3	2	2	2	2	7	3	1	1	4	0	2	1	1	1	1	1	1	3	1	17	12	1
42	74/M	2	1	2	2	2	2	3	1	1	1	1	2	1	1	1	1	1	1	2	1	6	5	6
43	48/M	3	1	1	1	2	1	3	1	1	11	2	1	3	3	2	2	1	2	1	1	15	1	2
44	31/M	3	2	2	2	1	1	3	1	1	1	2	1	1	1	1	1	1	1	1	1	5	3	1
45	39/M	3	1	1	1	2	1	3	1	1	11	2	2	1	1	2	2	1	2	1	1	1	1	2
46	17/M	3	2	2	2	3	1	3	1	1	1	0	1	1	1	1	1	1	1	1	1	1	8	1
47	67/M	3	1	1	1	1	3	3	1	1	3	2	4	1	1	1	1	1	1	1	2	3	1	3
48	65/M	5	1	1	1	2	1	3	1	1	2	2	1	1	2	1	1	1	1	4	2	3	1	2
49	44/M	2	1	1	1	1	3	3	2	1	1	2	1	1	1	1	1	1	1	1	1	3	1	3
50	34/F	3	1	1	1	1	1	2	1	1	1	0	1	1	1	1	1	3	1	1	1	14	1	2

SLN	A/S	SZR	FVR	HED	VMT	ALT	FND	PST	TRM	CSM	PI	PEH	BP	FUN	SUG	UR	Cr	SE	LFT	CXR	ECG	CT	MR	ETI
51	35/M	1	2	2	2	3	1	3	1	1	7	0	1	1	1	1	1	1	1	1	1	17	1	1
52	17/F	3	1	1	1	1	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	14	1	4
53	33/F	3	1	2	2	2	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	8	6	5
54	26/F	1	1	2	1	1	1	2	1	1	3	0	1	1	1	1	1	1	1	3	1	4	2	1
55	29/F	3	1	2	2	3	5	1	1	1	1	0	1	1	1	1	1	1	1	1	1	8	6	5
56	37/F	1	1	2	2	1	2	2	1	2	1	0	2	1	1	1	1	1	1	1	1	8	6	5
57	60/F	3	2	2	2	2	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	2	6	5
58	30/F	3	1	1	1	2	1	2	1	1	1	0	1	1	1	1	1	3	1	1	1	1	1	2
59	36/F	3	1	1	1	1	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	5	3	1
60	19/F	2	1	2	2	1	1	2	1	1	1	0	1	1	1	1	1	1	1	3	1	4	1	1
61	30/F	3	1	1	1	3	2	1	1	1	1	0	2	1	1	1	1	1	1	1	1	1	6	5
62	75/F	1	1	1	1	1	4	2	1	1	2	0	2	1	1	1	1	1	1	1	2	3	1	3
63	28/F	2	1	1	1	1	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	9	4	6
64	28/F	2	1	2	2	1	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	13	1	6
65	52/F	3	2	2	2	3	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	17	12	1
66	68/F	3	1	1	1	3	1	2	1	1	2	0	3	1	3	1	1	1	1	1	3	1	1	2
67	91/M	5	1	2	2	3	2	3	2	1	1	2	3	1	1	1	1	1	1	1	1	2	1	3
68	22/F	3	2	2	2	2	7	2	1	1	1	0	1	1	1	1	1	1	1	1	1	17	12	1
69	20/F	3	1	1	1	1	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	14	1	4
70	24/F	3	1	2	2	3	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	8	6	5
71	34/F	2	1	1	1	1	1	2	2	1	1	0	1	1	1	1	1	1	1	1	1	3	1	3
72	30/F	3	1	1	1	1	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	14	1	4
73	26/F	5	1	2	2	3	2	1	1	1	1	0	1	1	1	1	1	1	1	1	1	3	6	5
74	29/F	1	2	2	2	1	1	2	1	2	1	0	1	1	1	1	1	1	1	1	1	7	10	1
75	32/F	3	1	1	1	1	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	12	3	1

SL N	A/S	SZR	FVR	HED	VMT	ALT	FND	PST	TRM	CSM	PI	PEH	BP	FUN	SUG	UR	Cr	SE	LFT	CXR	ECG	CT	MR	ETI
76	50/M	3	1	2	1	2	1	3	1	1	2	0	1	1	3	2	2	1	1	1	3	1	1	2
77	56/M	1	1	2	2	1	2	3	1	1	1	2	2	1	1	1	1	1	1	2	1	6	1	6
78	40/M	1	2	2	1	2	1	3	1	1	1	2	2	1	1	1	1	1	1	1	1	3	1	3
79	31/M	3	2	1	1	2	1	3	1	1	2	0	1	1	3	2	2	1	1	1	1	1	1	2
80	24/F	5	1	2	2	3	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	6	5
81	27/F	1	1	2	1	1	1	2	1	1	1	0	1	1	1	1	1	1	1	1	1	14	1	4
82	44/F	5	1	2	2	3	2	2	1	1	1	0	1	1	1	2	2	1	1	1	1	1	2	5
83	20/F	5	2	2	2	2	4	2	1	1	1	0	1	1	1	1	1	1	1	1	1	3	12	1
84	14/F	5	2	2	2	3	7	2	1	1	1	0	1	1	1	1	1	1	1	1	1	17	8	1
85	55/M	1	1	1	1	1	1	3	1	1	1	2	1	1	1	1	1	1	1	1	1	13	1	6
86	72/M	3	1	2	2	1	3	3	1	1	1	2	2	1	1	1	1	1	1	2	1	12	1	6
87	24/M	5	1	1	1	2	2	3	1	2	1	0	1	1	1	1	1	1	1	1	1	3	6	5
88	58/M	3	1	1	1	1	1	3	1	1	2	1	2	1	2	2	2	1	1	4	3	1	1	2
89	45/M	3	1	1	1	3	7	3	1	1	7	2	1	1	1	1	1	1	1	3	1	1	12	1
90	55/M	1	1	1	1	1	1	3	1	1	1	2	2	1	1	1	1	1	1	1	3	3	1	3
91	65/M	5	1	1	1	3	1	3	1	1	9	2	2	1	1	2	2	1	1	1	2	1	1	2
92	55/M	1	1	1	1	1	3	3	1	1	3	2	2	1	1	1	1	1	1	1	3	3	1	3
93	35/M	1	1	2	1	3	3	3	1	1	1	0	4	1	1	1	1	1	1	1	1	2	1	3
94	70/M	5	1	1	2	3	1	3	1	1	1	3	2	1	1	1	1	2	1	1	1	1	1	2
95	28/M	5	2	2	2	3	1	3	1	1	1	0	1	1	1	1	1	1	1	1	1	1	8	1
96	64/M	3	1	1	1	2	1	3	1	1	2	2	2	1	2	1	1	1	1	1	1	1	1	2
97	74/M	1	1	1	1	1	3	3	1	1	3	1	4	1	1	1	1	1	1	1	1	2	1	3
98	60/M	3	1	1	1	2	1	3	1	1	9	0	4	1	1	3	3	1	1	4	2	1	1	2
99	65/M	5	1	1	1	2	1	3	1	1	3	2	4	1	1	3	3	1	1	4	2	1	1	2
100	24/M	1	1	1	1	1	1	3	1	1	1	0	1	1	1	1	1	1	1	1	1	13	7	7

KEY TO MASTER CHART

SZR(Seizures) 1- focal 2- focal – generalized 3- GTCS 4- EPC 5- Status epilepticus

FVR(Fever) 1 – absent, 2- present

HED(Headache) 1 – absent 2- present

VMT(Vomiting) 1 – absent 2- present

ALT(Altered sensorium) 1 – absent 2- drowsy 3- stupor /coma

FND(Focal neurological deficit) 1 –absent ,2- Right hemiplegia, 3-Left hemiplegia, 4- Monoplegia Right, 5- monoplegia Left, 6- Homonymous hemianopia 7-CN palsy

PST(Puerperal state) 1- Puerperal 2- Non puerperal 3- Not applicable

TRM(Trauma) 1-absent 2-present

CSM(CSOM) 1-absent 2-present

P I(Past illness) 1-None 2-DM 3-HTN 4-Tuberculosis 5-meningitis 6-malignancy 7-HIV positive 8-IHD/RHD 9-CKD 10-Old CVA 11- CLD

PEH(Personal history) 0-none 1-tobacco use 2-alcohol 3-contact with tuberculosis 4-poor sanitation

BP(Blood pressure) 1-normal 2-preHTN 3-stage 1 4-stage 2

FUN(Fundus) 1-normal 2-Retinopathy 3- Papilledema

SUG(Blood sugar) 1-normal 2-high 3-Low

UR(Blood urea) 1-normal 2-<90mg/dl 3->90mg/dl

Cr(Creatinine) 1- normal 2-<8mg/dl 3->8mg/dl

SE(S.Electrolytes) 1- normal 2-hyponatremia 3- hypocalcemia

LFT 1- normal 2-abnormal

CXR(Chest xray) 1-normal 2-mass 3-tuberculosis 4-cardiomegaly

ECG 1- normal 2-LVH 3-CAD

CT(CT scan) 1- normal 2-heamorhage 3-infarct
4-tuberculoma 5-neurocysticercosis 6-secondary
7-abcess 8-CVT 9-arachnoid cyst
10- osteomyelitis of bone with cerebritis 11-CSOM
12-ring enhancing lesion 13-tumour primary
14-calcification 15-Oedema 16-Atrophic brain
17- Dilated lateral ventricle

MR(MRI) 1-not done 2- tuberculoma 3- neurocysticercosis
4- tumour primary 5- secondary 6- CVT 7-AVM
8-encephalitis 9-heamangioma
10- abscess 11-Heamorrhage 12-meningitis